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Not Using Nose Clips: Another Thing To Worry About?

The Battle for Redesign

■ Are Nose Clips Necessary?

■ National Asthma Education Prevention Program Report II: Excerpts from the Internet

■ Points of View

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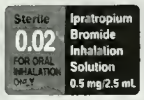

NEW!
Ipratropium Bromide
INHALATION SOLUTION

From DEY

Ipratropium Bromide Inhalation Solution 0.02%

BRIEF SUMMARY: Please see package insert for full prescribing information.

CONTRAINDICATIONS: Ipratropium bromide is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives.

WARNINGS: The use of Ipratropium Bromide Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation. Combination of Ipratropium Bromide Inhalation Solution and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

Immediate hypersensitivity reactions may occur after administration of ipratropium bromide, as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm and oropharyngeal edema.

PRECAUTIONS: General: Ipratropium bromide should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder neck obstruction.

Information for Patients: Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes. Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes. Patients should be advised that Ipratropium Bromide Inhalation Solution can be mixed in the nebulizer with albuterol or metaproterenol if used within one hour. Drug stability and safety of ipratropium bromide inhalation solution when mixed with other drugs in a nebulizer have not been established. Patients should be reminded that Ipratropium Bromide Inhalation Solution should be used consistently as prescribed throughout the course of therapy.

Drug Interactions: Ipratropium bromide has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators. Ipratropium bromide has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of ipratropium bromide.

Results of various mutagenicity studies (Ames test, mouse dominal lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative.

Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by ipratropium bromide administration. At doses above 90 mg/kg, increased resorption and decreased conception rates were observed.

Pregnancy: *TERATOGENIC EFFECTS:*
Pregnancy Category B Oral reproduction studies performed in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively and inhalation reproduction studies in rats and rabbits at doses of 1.5 and 1.8 mg/kg (or approximately 36 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of ipratropium bromide. However, no adequate or well controlled studies have been conducted in pregnant women. Because animal reproduction studies are not always predictive of human response, ipratropium bromide should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether ipratropium bromide is excreted in human milk. Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant to a significant extent, especially when taken by inhalation since ipratropium bromide is not well absorbed systemically after inhalation or oral administration. However, because many drugs are excreted in human milk, caution should be exercised when ipratropium bromide is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients below the age of 12 have not been established.

ADVERSE REACTIONS: Adverse reaction information concerning Ipratropium Bromide Inhalation Solution is derived from 12-week active-controlled clinical trials. Additional information is derived from foreign post-marketing experience and the published literature.

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table.

All Adverse Events, From a Double-blind, Parallel, 12-week Study of Patients with COPD*

	PERCENT OF PATIENTS				
	Ipratropium Bromide (500 mcg t.i.d.) n = 219	Meta-proterenol (15 mg t.i.d.) n = 212	Ipratropium / Bromide/ Metaproterenol (500 mcg t.i.d./ 15 mg t.i.d.) n = 108	Albuterol (2.5 mg t.i.d.) n = 205	Ipratropium Bromide/ Albuterol (500 mcg t.i.d./ 2.5 mg t.i.d.) n = 100
Body as a Whole-General Disorders					
Headache	6.4	5.2	6.5	6.3	9.0
Pain	4.1	3.3	0.9	2.9	5.0
Influenza-like symptoms	3.7	4.7	6.5	0.5	1.0
Back Pain	3.2	1.9	1.9	2.4	0.0
Chest Pain	3.2	4.2	5.6	2.0	1.0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0.9	1.9	0.9	1.5	4.0
Central & Peripheral Nervous System					
Dizziness	2.3	3.3	1.9	3.9	4.0
Insomnia	0.9	0.5	4.6	1.0	1.0
Tremor	0.9	7.1	8.3	1.0	0.0
Nervousness	0.5	4.7	6.5	1.0	1.0
Gastrointestinal System Disorders					
Mouth Dryness	3.2	0.0	1.9	2.0	3.0
Nausea	4.1	3.8	1.9	2.9	2.0
Constipation	0.9	0.0	3.7	1.0	1.0
Musculo-skeletal System Disorders					
Arthritis	0.9	1.4	0.9	0.5	3.0
Respiratory System Disorders (Lower)					
Coughing	4.6	8.0	6.5	5.4	6.0
Dyspnea	9.6	13.2	16.7	12.7	9.0
Bronchitis	14.6	24.5	15.7	16.6	20.0
Bronchospasm	2.3	2.8	4.6	5.4	5.0
Sputum Increased	1.4	1.4	4.6	3.4	0.0
Respiratory Disorder	0.0	6.1	6.5	2.0	4.0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13.2	11.3	9.3	12.2	16.0
Pharyngitis	3.7	4.2	5.6	2.9	4.0
Rhinitis	2.3	4.2	1.9	2.4	0.0
Sinusitis	2.3	2.8	0.9	5.4	4.0

*All adverse events, regardless of drug relationship, reported by the three percent or more patients in the 12-week controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with ipratropium bromide include tachycardia, palpitations, eye pain, urinary retention, urinary tract infection and urticaria. Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain have been reported.

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most common events leading to discontinuation of ipratropium bromide therapy in the 12-week trials. Headache, mouth dryness and aggravation of COPD symptoms are more common when the total daily dose of ipratropium bromide equals or exceeds 2,000 mcg.

Allergic-type reactions such as skin rash, angioedema of tongue, lips and face, urticaria, laryngospasm and anaphylactic reaction have been reported. Many of the patients had a history of allergies to other drugs and/or foods.

HOW SUPPLIED: Ipratropium Bromide Inhalation Solution is a clear, colorless solution supplied in a unit-dose vial containing 2.5 mL. Supplied in cartons as listed below:

25 vials per carton	NDC 49502-685-03
60 vials per carton	NDC 49502-685-60

Each vial is made from a low density polyethylene (LDPE) resin.

Store between 15° and 30°C (59° and 86°F). Protect from light. Retain in carton until time of use.

Caution: Federal law prohibits dispensing without prescription.

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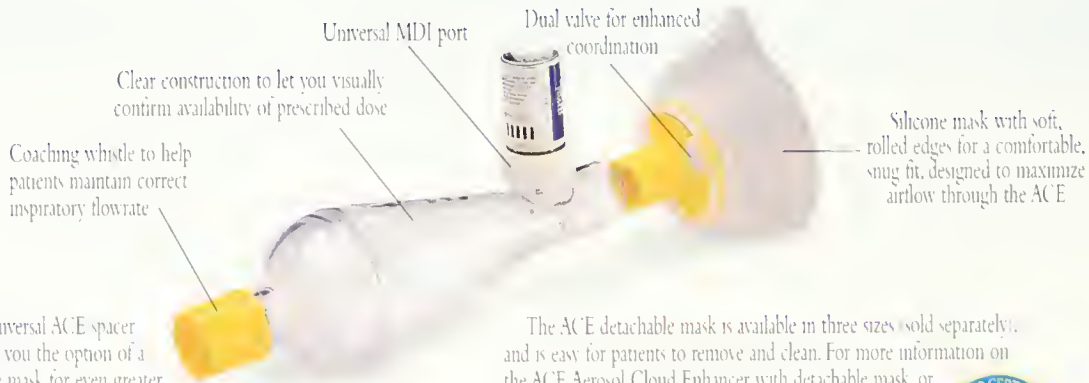
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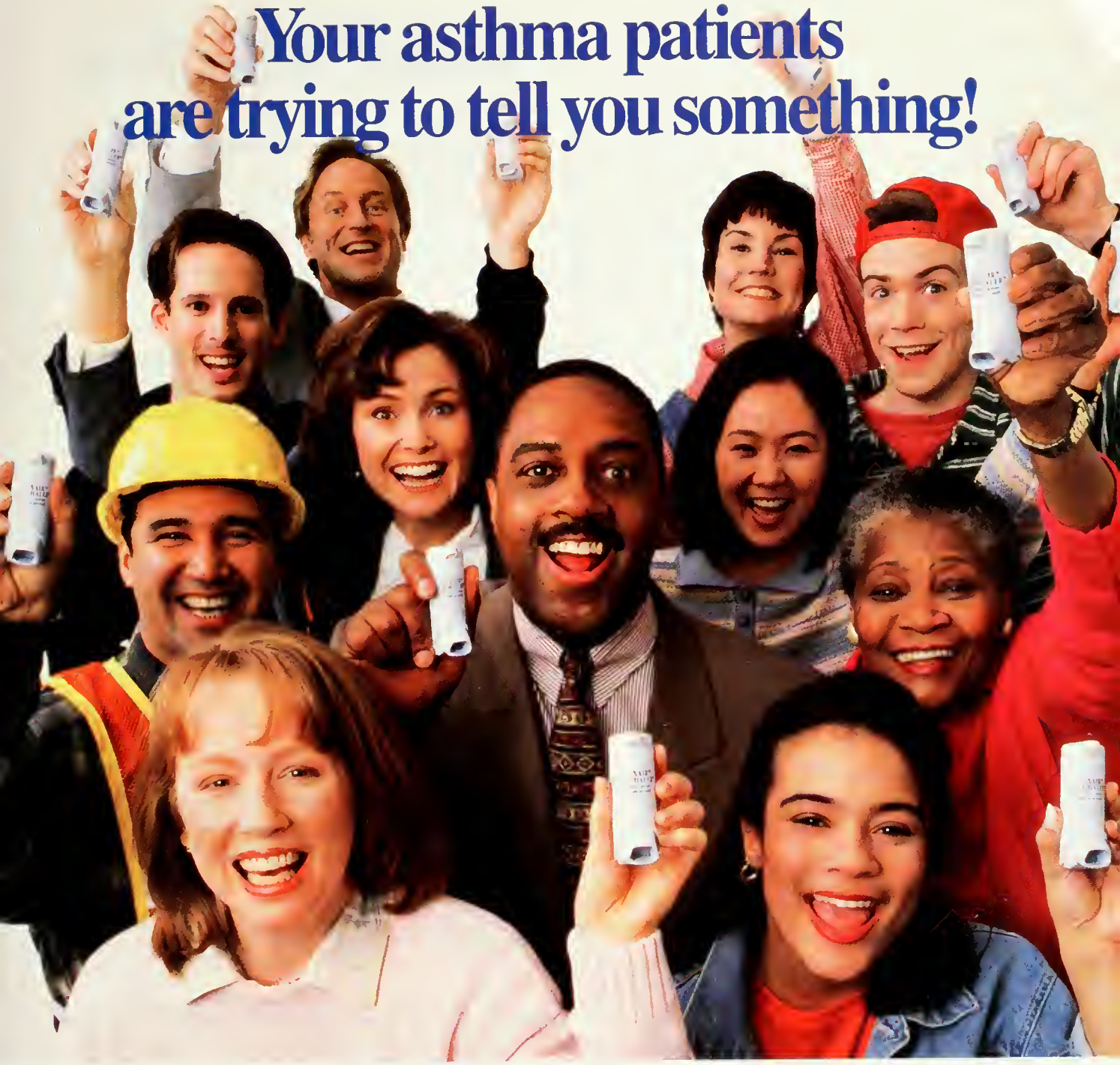

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Make it easier on your patients by prescribing the easier-to-use-correctly inhaler, Maxair Autohaler.

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* Data from a two-period, multi-dose, crossover trial conducted among 54 patients with chronic stable asthma who compared Maxair Autohaler with Ventolin®† press and breathe inhaler plus AeroChamber®‡. Percentages are patients' responses to end-of-study preference questions.

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Reference:
1. Study #1087, data on file, 3M Pharmaceuticals.

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(pirbuterol acetate inhalation aerosol) Bronchodilator Aerosol
For Inhalation Only
BRIEF SUMMARY

INDICATIONS AND USAGE. MAXAIR AUTOHALER is indicated for the prevention and reversal of bronchospasm in patients with reversible bronchospasm including asthma. It may be used with or without concurrent theophylline and/or steroid therapy.

CONTRAINDICATIONS: MAXAIR is contraindicated in patients with a history of hypersensitivity to any of its ingredients.

WARNINGS: As with other beta adrenergic aerosols, MAXAIR should not be used in excess. Controlled clinical studies and other clinical experience have shown that MAXAIR like other inhaled beta adrenergic agonists can produce a significant cardiovascular effect in some patients, as measured by pulse rate, blood pressure, symptoms, and/or ECG changes. As with other beta adrenergic aerosols, the potential for paradoxical bronchospasm (which can be life threatening) should be kept in mind. If it occurs, the preparation should be discontinued immediately and alternative therapy instituted.

Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs.

The contents of MAXAIR AUTOHALER are under pressure. Do not puncture. Do not use or store near heat or open flame. Exposure to temperature above 120 °F may cause bursting. Never throw container into fire or incinerator. Keep out of reach of children.

PRECAUTIONS: General. Since pirbuterol is a sympathomimetic amine, it should be used with caution in patients with cardiovascular disorders, including ischemic heart disease, hypertension, or cardiac arrhythmias, in patients with hyperthyroidism or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines or who have convulsive disorders. Significant changes in systolic and diastolic blood pressure could be expected to occur in some patients after use of any beta adrenergic aerosol bronchodilator.

Information for Patients. MAXAIR effects may last up to five hours or longer. It should not be used more often than recommended and the patient should not increase the number of inhalations or frequency of use without first asking the physician. If symptoms of asthma get worse, adverse reactions occur, or the patient does not respond to the usual dose, the patient should be instructed to contact the physician immediately. The patient should be advised to see the Illustrated Patient's Instructions for Use.

The Autohaler actuator should not be used with any other inhalation aerosol canister. In addition, canisters for use with MAXAIR AUTOHALER should not be utilized with any other actuator.

Drug Interactions. — Other beta adrenergic aerosol bronchodilators should not be used concomitantly with MAXAIR because they may have additive effects. Beta adrenergic agonists should be administered with caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, since the action of beta adrenergic agonists on the vascular system may be potentiated.

Carcinogenesis, Mutagenesis and Impairment of Fertility. — Pirbuterol hydrochloride administered in the diet to rats for 24 months and to mice for 18 months was free of carcinogenic activity at doses corresponding to 200 times the maximum human inhalation dose. In addition, the intragastric intubation of the drug at doses corresponding to 6250 times the maximum recommended human daily inhalation dose resulted in no increase in tumors in a 12-month rat study. Studies with pirbuterol revealed no evidence of mutagenesis. Reproduction studies in rats revealed no evidence of impaired fertility.

Teratogenic Effects — Pregnancy Category C. — Reproduction studies have been performed in rats and rabbits by the inhalation route at doses up to 12 times (rat) and 16 times (rabbit) the maximum human inhalation dose and have revealed no significant findings. Animal reproduction studies in rats at *oral* doses up to 300 mg/kg and in rabbits at oral doses up to 100 mg/kg have shown no adverse effect on reproductive behavior, fertility, litter size, peri- and postnatal viability or fetal development. In rabbits at the highest dose level given, 300 mg/kg, abortions and fetal mortality were observed. There are no adequate and well controlled studies in pregnant women and MAXAIR should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether MAXAIR is excreted in human milk. Therefore, MAXAIR should be used during nursing only if the potential benefit justifies the possible risk to the newborn.

Pediatric Use. MAXAIR AUTOHALER is not recommended for patients under the age of 12 years because of insufficient clinical data to establish safety and effectiveness.

ADVERSE REACTIONS: The following rates of adverse reactions to pirbuterol are based on single and multiple dose clinical trials involving 761 patients, 400 of whom received multiple doses (mean duration of treatment was 2.5 months and maximum was 19 months).

The following were the adverse reactions reported more frequently than 1 in 100 patients. **CNS:** nervousness (6.9%), tremor (6.0%), headache (2.0%), dizziness (1.2%). **Cardiovascular:** palpitations (1.7%), tachycardia (1.2%). **Respiratory:** cough (1.2%). **Gastrointestinal:** nausea (1.7%).

The following adverse reactions occurred less frequently than 1 in 100 patients and there may be a causal relationship with pirbuterol. **CNS:** depression, anxiety, confusion, insomnia, weakness, hyperkinesia, syncope. **Cardiovascular:** hypotension, skipped beats, chest pain. **Gastrointestinal:** dry mouth, glossitis, abdominal pain/cramps, anorexia, diarrhea, stomatitis, nausea and vomiting. **Ear, Nose and Throat:** smell/taste changes, sore throat. **Dermatological:** rash, pruritus. **Other:** numbness in extremities, alopecia, bruising, fatigue, edema, weight gain, flushing.

Other adverse reactions were reported with a frequency of less than 1 in 100 patients but a causal relationship between pirbuterol and the reaction could not be determined: migraine, productive cough, wheezing, and dermatitis.

The following rates of adverse reactions during three-month controlled clinical trials involving 310 patients are noted. The table does not include mild reactions.

PERCENT OF PATIENTS WITH MODERATE TO SEVERE ADVERSE REACTIONS

Reaction	Pirbuterol N=157	Metaproterenol N=153	Reaction	Pirbuterol N=157	Metaproterenol N=153
Central Nervous System			**Gastrointestinal**		
tremors	1.3%	3.3%	nausea	1.3%	2.0%
nervousness	4.5%	2.6%	diarrhea	1.3%	0.7%
headache	1.3%	2.0%	dry mouth	1.3%	1.3%
weakness	0%	1.3%	vomiting	0%	0.7%
drowsiness	0%	0.7%	**Dermatological**		
dizziness	0.6%	0%	skin reaction	0%	0.7%
Cardiovascular			rash	0%	1.3%
palpitations	1.3%	1.3%	**Other**		
tachycardia	1.3%	2.0%	bruising	0.6%	0%
Respiratory			smell/taste chg	0.6%	0%
chest pain/ tightness	1.3%	0%	backache	0%	0.7%
cough	0%	0.7%	fatigue	0%	0.7%
			hoarseness	0%	0.7%
			nasal congestion	0%	0.7%

OVERDOSAGE. The expected symptoms with overdosage are those of excessive beta-stimulation and/or any of the symptoms listed under adverse reactions, e.g., angina, hypertension or hypotension, arrhythmias, nervousness, headache, tremor, dry mouth, palpitation, nausea, dizziness, fatigue, malaise, and insomnia.

Treatment consists of discontinuation of pirbuterol together with appropriate symptomatic therapy.

The oral acute lethal dose in male and female rats and mice was greater than 2000 mg base/kg. The aerosol acute lethal dose was not determined.

Note: The indented statement below is required by the Federal government's Clean Air Act for all products containing or manufactured with chlorofluorocarbons (CFC's):

WARNING: Contains trichloromonofluoromethane and dichlorodifluoromethane, substances which harm public health and environment by destroying ozone in the upper atmosphere.

A notice similar to the above WARNING has been placed in the "Patient's Instructions for Use" of this product pursuant to EPA regulations.

CAUTION: Federal law prohibits dispensing without prescription.
Store between 15° and 30° C (59° to 86° F).
For Full Prescribing Information see package insert.

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Editorials, Commentaries, and Reviews To Note

Proceedings from the First International Symposium on the Congenital Central Hypoventilation Syndrome Held in New Orleans, LA, on May 11, 1996—D Gozal, C Gaultier. Pediatr Pulmonol 1997;23:133-168.

Lung and Heart-Lung Transplantation in Children (State of the Art)—BE Noyes, G Kurland, DM Orenstein. Pediatr Pulmonol 1997;23:39-48.

Usefulness of Flow Volume Loops in Emergency Center and ICU Settings (Pulmonary Physiologic Test of the Month)—KK Guntupalli, V Bandi, C Sirgi, C Pope, A Rios, W Eschenbacher. Chest 1997;111(2):481-488.

Pulmonary Artery Flotation Catheters: A Statement by the American College of Chest Physicians and the American Thoracic Society (Editorial)—B Chernow. Chest 1997;111(2):261.

Asthma Management: Perspectives and Paradigms in a Changing Environment (Supplement)—A Rebuck, PM O'Byrne, Conference Chairs. Chest, 1997;111(2):1S-68S.

Medical Literature Made Easy: Querying Databases on the Internet—R Sikorski, R Peters. JAMA 1997;277(12):959-960.

Prone Position in Mechanically Ventilated Patients with Severe Acute Respiratory Failure—G Chatte, J-M Sab, J-M DuBois, M Sirodot, P Gaussorgues, D Robert. Am J Respir Crit Care Med 1997;155:473.

The purpose of this study was to characterize changes in oxygenation, expressed as P_{aO_2}/F_{IO_2}, when patients with severe acute respiratory failure ($P_{aO_2}/F_{IO_2} < 150$), unrelated to left ventricular failure or atelectasis, were turned to and from a supine to prone position at 1- and 4-hour intervals. Ventilator settings were unchanged. Thirty-two consecutive patients were studied 1 hour before, 1 and 4 hours during, and 1 hour after placing in a prone position with P_{aO_2}/F_{IO_2} of 103 ± 28, 158 ± 62, 159 ± 59, and 128 ± 52, respectively (ANOVA, $p < 0.001$). After 1 hour in a prone position, improvement of P_{aO_2}/F_{IO_2} by 20 mm Hg or more was considered a positive response. Seven patients studied had no response (22%), hereafter referred to as nonresponders, and 25 had a positive response (78%), hereafter referred to as responders. Among the 7 nonresponders, 2 did not tolerate the prone position and were returned supine before the end of the 4-hour trial. With the remaining 5, P_{aO_2}/F_{IO_2} evolution was 83 ± 29, 77 ± 19, 83 ± 33, and 81 ± 47, respectively. For 2 of the 25 responders, measurements are missing after returning to the supine position. In 10 of the 23 responders (43%) who completed the 4-hour prone trial, the P_{aO_2}/F_{IO_2} returned to its starting value when patients were repositioned supine: 117 ± 24, 164 ± 44, 156 ± 55, and 110 ± 34, respectively (ANOVA, $p < 0.01$). In 13 of the 23 (57%) improvement persisted: 105 ± 27, 187 ± 58, 189 ± 49, and 157 ± 49, respectively (ANOVA, $p < 0.001$). Repeated improvements after turning to a prone position were frequently observed. Side effects in the 32 patients after a total of 294 periods in a prone position included minor skin injury and edema, 2 instances of apical atelectasis, 1 catheter removal, 1 catheter compression, 1 extubation, and 1 transient supraventricular tachycardia. *See the related editorial:* ***For Every Thing (Turn ... Turn ... Turn....)****—RK Albert. Am J Respir Crit Care Med 1997;155:393-394.*

Observations on the Effects of Aerosolized Albuterol in Acute Asthma—L Strauss, R Hejal, G Galan, L Dixon, ER McFadden Jr. Am J Respir Crit Care Med 1997;155:454.

To determine the dose of albuterol required to terminate acute episodes of asthma, 92 acutely ill subjects received 3 doses of 2.5 mg each by nebulization every 20 min. Peak expiratory flow rates (PEFR) and signs and symptoms were serially monitored. A dose-response increase in pulmonary function was found, but only 66% of the subjects improved sufficiently to be sent home. Of these, 56% required ≤ 5.0 mg of drug to reach the discharge threshold, whereas the remainder needed 7.5 mg. In 34% of participants, albuterol was ineffectual. These individuals were characterized by more severe obstruction at presentation, and after 3 doses of medication their PEFR still did not exceed 40% of the expected value. Further treatment in the emergency department (ED) or hospital was not immediately helpful, and these patients ultimately required 3.8 ± 0.4 days of inpatient care to become asymptomatic. There were no discernible differences between responders and nonresponders in the type or quantity of medications used. However, the nonresponders had more severe disease as measured by recurrent hospitalizations and ED visits. This study demonstrates that, in emergency situations, albuterol does not relieve acute airway obstruction in all asthmatic individuals with equal efficacy. Two-thirds of patients are sensitive, and in these patients 5 to 7.5 mg of albuterol provides optimal treatment. In the remainder, albuterol, even in high doses, has little effect for days.

Pulmonary Sequelae in Infants Treated with Extracorporeal Membrane Oxygenation—JS Greenspan, MJ Antunes, WJ Holt, D McElwee, JA Cullen, AR Spitzer. Pediatr Pulmonol 1997; 23:31.

The decision to place an infant on extracorporeal membrane oxygenation (ECMO) is based

on predictions of expected morbidity and mortality. One unknown factor is the relationship between pre-ECMO pulmonary dysfunction and on barotrauma and post-ECMO pulmonary sequelae. To determine whether placement of infants on extracorporeal membrane oxygenation (ECMO) *early* is associated with less subsequent pulmonary dysfunction than placing infants on ECMO *later*, we evaluated pulmonary function in 25 neonates prior to ECMO, when the infants had come off ECMO, and at the time of nursery discharge. Pulmonary resistance (R) and compliance (C_L) were determined by a pneumotachograph and esophageal manometry, and functional residual capacity (FRC) was determined by a helium dilution method. Maximal expiratory flow (V̇maxFRC) was determined by thoracic compression at the time of discharge. Infants were assigned to an *early* ECMO group (< 36 hours of age, n = 12), or a *late* ECMO group (> 36 hours of age, n = 13). When first evaluated, the *early* group had a higher oxygenation index than the *late* group (mean value, 63 vs 48), but *initial* pulmonary function measurements were not different between the 2 groups. In the *early* group mean C_L increase from 0.20 to 0.36 mL/cm H_2O/kg, FRC increased from 7 to 20 mL/kg, and mean R decreased from 107 to 61 cm H_2O/L/s between the initial study and immediately after ECMO. In the *late* group, only FRC increased from a mean of 8 to 20 mL/kg. C_L and FRC increased from post-ECMO to discharge in both groups (mean C_L from 0.36 to 0.76 mL/cm H_2O/kg in the *early* group, and from 0.30 to 0.79 in the *late* group). Mean FRC increased from 20 to 26 mL/kg in the *early* group, and from 20 to 25 mL/kg in the *late* group. V̇maxFRC was lower in the *late* than the *early* group at discharge (mean, 1.14 vs 1.58 L/s; $p < 0.05$). While both groups of infants had minimal pulmonary dysfunction at discharge, the infants placed on ECMO *early* had evidence of slightly *less* airway dysfunction despite a higher initial oxygenation index than the infants placed on ECMO late.

Ventilatory and Hemodynamic Effects of Continuous Positive Airway Pressure in Left Heart Failure—F Lenique, M Habis, F Lofaso, J-L Dubois-Randé, A Hart, L Brochard. Am J Respir Crit Care Med 1997;155:500.

The ventilatory and hemodynamic effects of continuous positive airway pressure (CPAP) delivered via a face mask (at 0, 5, and 10 cm H_2O, and after a return to 0 cm H_2O) were studied in 9 patients with acute left heart failure (pulmonary artery occlusion pressure [PAOP] ≥ 18 mm Hg, and cardiac index [CI] ≤ 2.8 L/min/m^2). CPAP at 10 cm H_2O induced an improvement in lung compliance (60 ± 10 mL/cm H_2O to 87 ± 20 mL/cm H_2O, $p < 0.05$) and in lung and airway resistance (5.7 ± 1.0 cm H_2O/L/s to 3.4 ± 1.0 cm H_2O/L/s, $p < 0.05$), a reduction in work of breathing (18 ± 3 J/min to 12 ± 2 J/min, $p < 0.05$), and in the pressure-time index of the respiratory muscles (279 ± 22 cm H_2O/s/min to 174 ± 25 cm H_2O/s/min, $p < 0.05$), without significant changes in breathing pattern. Despite a significant reduction in the negative swings in intrathoracic pressure (15.2 ± 1.9 cm H_2O to 10.8 ± 1.8 cm H_2O, $p < 0.001$), no significant change was observed in CI or stroke volume during CPAP. However, mean transmural filling pressures decreased significantly with CPAP, suggesting a better cardiac performance. Neither the level of stroke volume nor of PAOP, was predictive of changes in CI or in stroke volume. In patients with respiratory insufficiency caused by congestive heart failure (CHF), CPAP reduces respiratory muscle effort without altering cardiac output. The slight decrease in mean transmural left and right atrial pressures suggests an improvement in cardiac performance.

Perceived Control of Asthma: Development and Validation of a Questionnaire—PP Katz, EH Yelin, S Smith, PD Blanc. Am J Respir Crit Care Med 1997;155:577.

Psychological factors can play a role in asthma symptoms and may play a role in how individuals manage asthma. Because poor self-management of asthma has been linked to poor outcomes, it is important to understand perceived control of asthma—the individual's perceived ability to deal with asthma and its exacerbations effectively. This study used data from an ongoing panel study of adults with asthma (n = 601). The 11-item Perceived Control of Asthma Questionnaire (PCAQ) demonstrated internal consistency (Cronbach's alpha = 0.74) and excellent construct validity, correlating strongly with asthma severity, quality of life, and Medical Outcomes Study Short Form (SF-36) measures of health status ($p < 0.05$). After controlling for demographics and asthma severity, each 6-point decrement in PCAQ score was significantly associated with increased risk of hospitalization (OR = 1.4 [95% CI: 1.1-1.8]), frequent activity restriction (OR = 1.5 [1.2-1.8]), and, among those with labor force participation (n = 551), asthma-related cessation of employment (OR = 1.7 [1.1-2.4]). The PCAQ is a short, easy-to-administer, reliable, and valid measure of perceived control of asthma. It is strongly associated with adverse asthma outcomes even taking into account demographic characteristics and asthma severity, suggesting that patient-centered interventions focusing on perceived control might improve asthma outcomes.

Determinants of Lung Volume in Spontaneously Breathing Preterm Infants—CF Poets, GA Rau, K Neuber, M Gappa, J Seidenberg. Am J Respir Crit Care Med 1997;155:649.

To study the effects of apneic pauses, sighs, and breathing patterns on functional residual capacity (FRC), we measured FRC repeatedly in 48 healthy preterm infants (weight at study 2,042 ± 316 g [mean ± SD], postconceptional age 36.6 ± 2.0 wk), during unsedated sleep using a modified heliox/nitrogen washout technique. Breathing movements and pulse oximeter saturation (S_{pO_2}) were recorded throughout and recordings analyzed for the presence of regular and nonregular breathing pattern, apneic pauses, sighs, and desaturations ($S_{pO_2} \leq 90\%$) during the last 2 minutes prior to each FRC measurement. FRC was lower during nonregular than during regular breathing pattern (23.3 ± 7.2 mL/kg vs 26.9 ± 7.8 mL/kg, $p < 0.02$); however, this apparent effect of breathing pattern disappeared after controlling the data for apneic pauses. Apneic pauses resulted in a significant decrease in FRC: mean FRC was 20.0 ± 6.8 mL/kg if measured within 2 minutes of an apneic pause, 26.0 ± 6.9 mL/kg if measured after a sigh ($p < 0.001$), and 24.0 ± 7.7 mL/kg if there had been neither a sigh nor an apneic pause ($p < 0.05$). The interval between the apneic pause and the FRC measurement had no effect on FRC. There was an inverse correlation between FRC and the speed with which S_{pO_2} fell during desaturation ($r = -0.5$, $p < 0.03$). Apneic pauses resulted in a persistent reduction in FRC in these preterm infants. Sighs appeared to restore FRC. The significant relationship between FRC and the speed of desaturation found in this study underscores the importance of endogenous or exogenous strategies that help to increase FRC, such as sighs or the application of continuous positive airway pressure, for the stability of oxygenation in preterm infants who have difficulty maintaining their oxygenation.

Combined Inhalation of Nitric Oxide and Oxygen in Chronic Obstructive Pulmonary Disease—M Yoshida, O Taguchi, EC Gabazza, T Kobayashi, T Yamakami, H Kobayashi, K Maruyama, T Shima. Am J Respir Crit Care Med 1997;155:526.

Inhaled nitric oxide (NO) has been shown to reduce the mean pulmonary artery pressure (mPAP) and to improve P_{aO_2} in patients with acute respiratory failure undergoing oxygen (O_2) therapy. However, inhaled NO reduced pulmonary hypertension without improving P_{aO_2} in patients with chronic obstructive pulmonary disease (COPD). This study was intended to compare the hemodynamic and gas exchange responses during inhalation of NO or O_2 with those observed during the combined inhalation of NO and O_2 in 10 spontaneously breathing COPD patients. Hemodynamic and blood gas parameters were measured after breathing: (1) room air; (2) NO added to room air; (3) O_2 (1 L/min); or (4) NO and O_2. During inhalation of 2 ppm NO added to room air, the mPAP (23.1 ± 2.5 vs 20.6 ± 2.2 mm Hg) and the pulmonary vascular resistance (PVR) (434 ± 70 vs 378 ± 50 dyne s/cm^5) were significantly ($p < 0.05$) lower than those measured with room air. However, the values of P_{aO_2} did not improve. The

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combined inhalation of NO and O_2 was associated not only with a significant ($p < 0.05$) decrease of mPAP (21.4 ± 2.3 vs 19.7 ± 1.8 mm Hg) and PVR (431 ± 72 vs 370 ± 44 dyne s/cm^5), but also with a remarkable improvement ($p < 0.05$) in the values of P_{aO_2} (91.4 ± 6.6 vs 111.5 ± 7.8 mm Hg) as compared with values obtained during the inhalation of O_2 alone. These findings suggest that combined therapy with NO and O_2 may constitute an alternative approach to treating patients with COPD and pulmonary hypertension.

Summertime Haze Air Pollution and Children with Asthma—GD Thurston, M Lippmann, MB Scott, JM Fine. Am J Respir Crit Care Med 1997; 155:654.

In order to investigate associations between summertime haze air pollution and asthma at an individual level, 52, 58, and 56 children (ages 7-13) attending a summer "asthma camp" were followed during the last week of June in 1991, 1992, and 1993, respectively. Most of the subjects had moderate to severe asthma. Daily records were kept of the environmental conditions, as well as of subject medication use, lung function, and medical symptoms. Air pollution was found to be significantly and consistently correlated with acute asthma exacerbations, chest symptoms, and lung function decrements. The pollutant most consistently associated with adverse health consequences was ozone (O_3), although associations with sulfates and hydrogen ion suggest a possible role by fine particles as well. Effects were found to be roughly monotonic as a function of O_3 concentration. Regression of morning (8:00 AM) to afternoon (5:00 PM) peak flow change on O_3 indicated pulmonary function reductions similar to those previously reported for more active children without asthma. Moreover, analyses also indicated an increased risk of an asthma exacerbation and of experiencing chest symptoms of approximately 40% on the highest pollution day, relative to the mean. Based on these relative risk estimates, a rise in the 1-hour daily maximal O_3 from 84 ppb to 160 ppb was associated in this group with an increase from 20 to 28 (± 2) in the expected number of unscheduled medications administered/day, and from 29 to 41 (± 3) in the expected total number of chest symptoms/day. Thus, air pollution can be a major contributor to the respiratory problems experienced by children with asthma during the summer months.

Childhood Asthma and Lung Function in Mid-Adult Life—H Oswald, PD Phelan, A Lanigan, M Hibbert, JB Carlin, G Bowes, A Olinsky. Pediatr Pulmonol 1997;23:14.

The longitudinal lung function data in 286 subjects from a 28-year follow-up of childhood asthma is reported. Airway obstruction in mid-adult life was present mainly in those with moderately severe asthma. Subjects who had been wheeze free for at least 3 years, even if asthma had been persistent in childhood, had normal lung function and no increased bronchial reactivity. Only 2 subjects, both with persistent asthma from childhood, failed to show an improvement in FEV_1 of greater than 10% following inhalation of a β-adrenergic agonist. Subjects with relatively mild asthma who had not taken inhaled steroids did not appear to be disadvantaged with respect to lung function.

Inhaled and Nasal Glucocorticoids and the Risks of Ocular Hypertension or Open-Angle Glaucoma—E Garbe, J LeLorier, J-F Boivin, S Suissa. JAMA 1997;277(9):722.

OBJECTIVE: To determine whether the use of inhaled and nasal glucocorticoids is associated with an increased risk of ocular hypertension or open-angle glaucoma. DESIGN: Case-control study. SETTING: Québec universal health insurance program for all elderly (registered in the Régie de l'assurance maladie du Québec [RAMQ] database). PATIENTS: Enrollees in the RAMQ database aged 66 years and older. The 9,793 case patients were ophthalmology patients with a new diagnosis of borderline glaucoma or open-angle glaucoma or were newly started on treatment for ocular hypertension or glaucoma between 1988 and 1994. The 38,325 control patients were randomly selected among noncases with ophthalmologist visits in the same month and year as the case event (index date). MAIN OUTCOME MEASURES: The odds ratio (OR) of ocular hypertension or open-angle glaucoma was determined in patients using inhaled or nasal glucocorticoids relative to nonusers, using conditional logistic regression analysis. The OR was adjusted for age, sex, diabetes mellitus, systemic hypertension, use of ophthalmic and oral glucocorticoids, and characteristics of health care system use in the year before the index date. RESULTS: Overall, current use of inhaled and nasal glucocorticoids was not associated with an increased risk of ocular hypertension or open-angle glaucoma. Current users of high doses of inhaled steroids prescribed regularly for 3 or more months were at an increased risk with an OR of 1.44 (95% confidence interval, 1.01-2.06). CONCLUSION: Prolonged administration of high doses of inhaled glucocorticoids increases the risk of ocular hypertension or open-angle glaucoma. This finding suggests that in these patients intraocular pressure monitoring may be warranted.

A Prospective Study of Risk Factors for Pulmonary Embolism in Women—SZ Goldhaber, F Grodstein, MJ Stampfer, JE Manson, GA Colditz, FE Speizer, WC Willett, CH Hennekens. JAMA 1997;277(8):642.

OBJECTIVE: To investigate risk factors for pulmonary embolism in women. DESIGN: Prospective study based on biennial, mailed questionnaires. SETTING: Nurses' Health Study with 16 years of follow-up from 1976 to 1992. PATIENTS: A group of 112,822 women aged 30 to 55 years in 1976, free from diagnosed cardiovascular disease or cancer at baseline. Overall, there were 1,619,770 person-years of follow-up. MEASUREMENTS: Based on self-report and medical records, we documented 280 cases of pulmonary embolism, of which 125 were primary (no identified antecedent cancer, trauma, surgery, or immobilization). Information on height, weight, cigarette smoking, hypertension, diabetes, and hypercholesterolemia was collected by questionnaire. RESULTS: In multivariate analysis, obesity, cigarette smoking, and hypertension were independent predictors of pulmonary embolism. Specifically, obese women (body mass index ≥ 29.0 kg/m2) had an increased risk of primary pulmonary embolism (multivariate relative risk = 2.9; 95% confidence interval [CI], 1.5-5.4). Heavy cigarette smokers also had an increased risk of primary pulmonary embolism. The relative risk (RR) of primary pulmonary embolism was 1.9 (95% CI, 0.9-3.7) for women currently smoking 25 to 34 cigarettes/day and 3.3 (95% CI, 1.7-6.5) for those smoking 35 cigarettes or more daily as compared with never smokers. Hypertension, even after adjustment for body mass index, was also associated with an increased risk of primary pulmonary embolism (RR = 1.9; 95% CI, 1.2-2.8). High serum cholesterol levels (RR = 1.1; 95% CI, 0.62-1.8) and diabetes (RR = 0.7; 95% CI, 0.3-1.9) did not appear to be related to primary pulmonary embolism. CONCLUSION: These prospective data indicate that obesity, cigarette smoking, and hypertension are associated with increased risk of pulmonary embolism in women. Control of these risk factors will decrease risks of pulmonary embolism as well as coronary heart disease.

Relationship of Microbiologic Diagnostic Criteria to Morbidity and Mortality in Patients with Ventilator-Associated Pneumonia—F Bregeon, L Papazian, A Visconti, R Gregoire, X Thirion, F Gouin. JAMA 1997;277(8):655.

OBJECTIVE: To evaluate whether the mortality and the morbidity of ventilator-associated pneumonia, defined by positive result of protected specimen brush culture, was different from that defined by other methods. DESIGN: Matched-cohort study. All patients with clinical suspicion of pneumonia were investigated with protected specimen brush, bronchoalveolar lavage, and blind bronchial samplings. Two groups were defined: brush-positive patients (positive culture of the protected specimen brush) and brush-negative patients (negative culture of the protected specimen brush, but positive culture with another method). SETTING: A 14-bed medicosurgical intensive care unit (ICU) in an 850-bed teaching hospital. PATIENTS: All patients with documented ventilator-associated pneumonia over 4 years 9 months. A total of 162 cases documented by protected specimen brush

culture and 223 documented by another sampling procedure. Patients were matched according to diagnosis on admission, age, sex, date of admission, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and date of onset of pneumonia. MAIN OUTCOME MEASURES: Mortality rate, duration of mechanical ventilation, duration of ICU stay, duration of hospital stay, sampling methods, and microbiologic cultures. RESULTS: A total of 76 pairs were submitted for analysis. The effectiveness of matching was 81.85%. There was no difference in mortality between brush-positive patients and brush-negative patients. The ICU fatality rate was 38% in the brush-positive group and 39.4% in the brush-negative group (odds ratio [OR], 0.95; 95% confidence interval [CI], 0.46-1.93). The hospital fatality rate was 41% (OR, 1; 95% CI, 0.5-2.01). The mean (SD) duration of ventilation was 26 (23) days in the 2 groups (range, 3-132 days). The duration of ICU stay was 33 (27.4) days in the 2 groups (range, 3-152 days). CONCLUSIONS: When confounding factors are controlled, patient outcome is the same if ventilator-associated pneumonia has been diagnosed by protected specimen brush or by another sampling method.

Airway Function Tests and Vocal Cord Paralysis in Lung Transplant Recipients—A Zapletal, G Kurland, SR Boas, BE Noyes, P Greally, A Faro, JM Armitage, DM Orenstein. Pediatr Pulmonol 1997;23:87.

Maximum expiratory and inspiratory flow-volume (MEFV, MIFV) curves, specific airway conductance (sG_{aw}), and flexible fiberoptic laryngoscopy were examined in 8 pediatric lung transplant recipients with vocal cord paralysis (VCP). Six were heart-lung (H-L) and 2 double-lung (D-L) recipients, 7 had left VCP, and 1 had right VCP. Based on the pulmonary function tests (PFT), 2 subgroups could be distinguished in the 8 recipients with VCP. Group A (5/8 recipients; mean age, 13 ± 3.4 years; mean height, 144.3 ± 12.3 cm) had significantly reduced specific airway conductance (sG_{aw}; < 2 SD from predicted) and normal MEF_{25}, MEF_{50}, peak expiratory flow (PEF), forced expiratory volume in 1 second (FEV_1), and $\%FEV_1$/forced vital capacity (FVC); this pattern suggested variable extrathoracic airway obstruction. PIF was normal in 4/5 and reduced in 1/5 of these recipients. Group B (3/8 recipients with VCP; mean age, 17 ± 2.4 years; mean height, 156.3 ± 12.0 cm) had significantly reduced sG_{aw}, MEF_{25}, MEF_{50}, PEF, FEV_1, and $\%FEV_1$/FVC, implying primarily small airway obstruction. These recipients had bronchiolitis obliterans. The results suggest that a pattern of reduced sG_{aw} and normal MEFs, PEF, FEV_1, and PIF should raise the possibility of VCP in patients after lung transplantation. sG_{aw} is more sensitive than PIF and PEF in identifying airway obstruction due to VCP, and should be routinely included in the follow-up evaluation of lung transplant recipients.

Bedside Methods for Detecting Aspiration in Tube-Fed Patients—NA Metheny, RE Clouse. Chest 1997;111(3):724.

This literature review compares the efficacy of the 2 most commonly recommended bedside tests for detecting aspiration in tube-fed patients: (1) adding dye to the formula and observing for its appearance in tracheobronchial secretions, and (2) using glucose oxidase reagent strips to test tracheobronchial secretions for glucose-containing enteral formula. The review also describes potential problems and cost associated with each test. Several studies indicate that the dye method is far less sen-

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sitive than the glucose reagent method: further, there are anecdotal reports of potential harm to patients when dye is added to tube feedings. Specificity of the glucose method has been challenged by several authors; also, cost is probably greater with this method.

Adverse Effects of Corticosteroid Therapy for COPD: A Critical Review—CE McEvoy, DE Niewoehner. Chest 1997;111(3):732.

Inhaled and systemic corticosteroids are commonly prescribed for the treatment of COPD. Despite their frequent use, there is insufficient evidence regarding efficacy of steroid therapy in COPD. While awaiting the results of more definitive prospective trials, the clinician must evaluate whether the benefits of such therapy outweigh the potential for adverse events. This is particularly pertinent in the population of patients with COPD who generally are older, less active, and have significant tobacco-use histories, all of which may place them at greater risk for adverse effects. In this review, we examine the current scientific evidence supporting the many purported adverse systemic effects associated with the use of corticosteroids in the treatment of COPD.

Early Complications and Value of Initial Clinical and Paraclinical Observations in Victims of Smoke Inhalation without Burns—P Hantson, R Butera, J-L Clemessy, A Michel, FJ Baud. Chest 1997;111(3):671.

OBJECTIVE: To evaluate the incidence of early pulmonary complications and the value of initial clinical signs and paraclinical investigations in victims of smoke inhalation not suffering from burns following structural fires. DESIGN: Retrospective chart review. SETTING: Thirteen-bed ICU. PATIENTS: Sixty-four victims of smoke inhalation following household fires were admitted to the ICU between January 1987 and December 1992. Exclusion criteria from the study were patients with cutaneous burns or multiple trauma or blast injury, and patients found in cardiac arrest. METHODS: Clinical, biological, and radiologic parameters were collected over a 5-day period. RESULTS: The mortality rate in relation to progressive respiratory failure was 3.1%. Mean ICU stay was 5.8 days (range, 1 to 33 days), and was longer in the patients presenting with soot deposits in the oropharynx ($p = 0.02$), dysphonia (D) ($p = 0.05$), or ronchi (R) ($p = 0.0004$) at the first examination, and in those having a positive sputum bacteriologic analysis ($p = 0.003$) or requiring parenteral bronchodilator agents for more than 24 hours ($p = 0.04$). Thirty-five patients underwent mechanical ventilation (MV) for a mean of 101.2 hours (range, 8 to 648 h). Mean MV duration was higher in the patients presenting initially with R ($p = 0.003$), high carbon monoxide (but not cyanide) levels ($p = 0.02$), or a positive bacteriologic sample ($p = 0.0001$). Positive bacteriologic sampling correlated with the presence of D ($p = 0.02$) or R ($p = 0.04$) and with immediate intubation ($p = 0.0003$). No correlation was found with chest radiograph. CONCLUSIONS: In this selected series of fire victims without cutaneous burns, respiratory injury was frequent. The initial clinical signs may be helpful to predict pulmonary complications.

Predictors of Postoperative Pulmonary Complications Following Abdominal Surgery—JA Brooks-Brunn. Chest 1997;111(3):564.

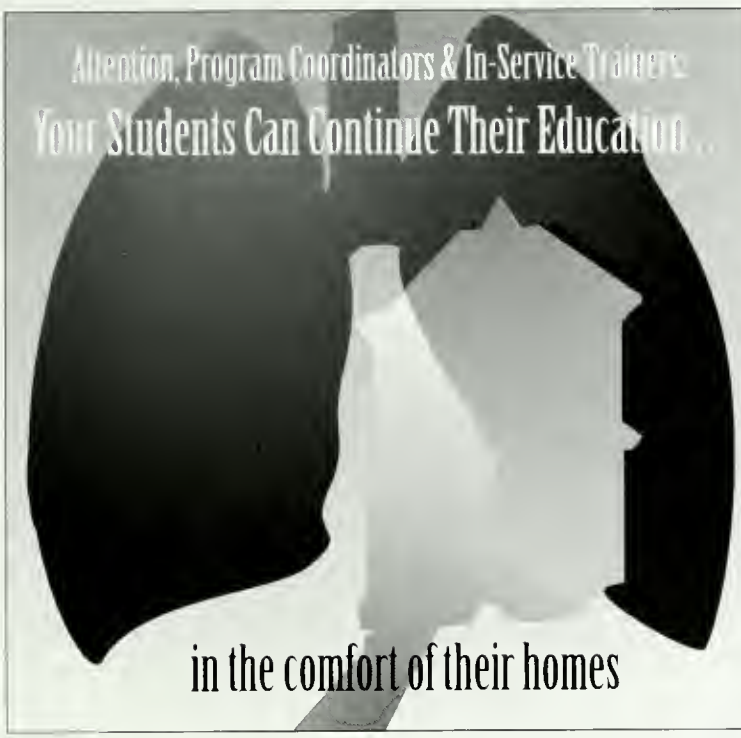
OBJECTIVE: To determine how risk factors could be combined to best predict the development of a postoperative pulmonary complication (PPC) following abdominal surgery. DESIGN: Prospective model-building study. Logistic regression models were developed using significant risk factors identified in the univariate analysis. SETTING: Four midwestern hospitals. PATIENTS: Convenience sample of 400 patients who underwent abdominal surgical procedures between January 1993 and August 1995. MEASUREMENTS & RESULTS: Multicriteria outcome for postoperative pulmonary complication used to collectively assess atelectasis and pneumonia. Twenty-three risk factors were assessed. Six risk factors were identified as independent by logistic regression: age ≥ 60 years (adjusted odds ratio [Adj OR], 1.89); impaired preoperative cognitive function (Adj OR, 5.93); smoking history within the past 8 weeks (Adj OR, 2.27); body mass index ≥ 27 (Adj OR, 2.82); history of cancer (Adj OR, 2.23); and incision site-upper abdominal or both upper/lower abdominal incision (Adj OR 2.30). CONCLUSIONS: These results provide a framework for identifying patients at risk of developing a PPC following abdominal surgery. A reliable and valid risk index could be used clinically to guide preoperative and postoperative pulmonary care and target-limited resources for patients at risk.

Incentive Spirometry Performance: A Reliable Indicator of Pulmonary Function in the Early Postoperative Period after Lobectomy?—R Bastin, J-J Moraine, G Bardocsky, R-J Kahn, C Mélot. Chest 1997;111(3):559.

OBJECTIVES: The purpose of our study was to validate the incentive spirometry (IS) as a simple mean to follow pulmonary function at the bedside after lung surgery. MATERIALS & METHODS: We studied prospectively 19 patients (16 men, 3 women; mean ± SE age, 60 ± 2.8 yr) undergoing lobectomy for lung cancer. All the patients had an obstructive pattern with FEV_1/FVC below 75%. Lung volumes, including functional residual capacity (FRC) and residual volume (RV), measured using spirometry and the helium dilution technique, and IS were measured preoperatively and postoperatively at Days 1, 2, 3, and 8, and at 2 months. RESULTS: Our results showed that in the postoperative period after lung resection, IS performance was well correlated (R) during the first 8 postoperative days with vital capacity (VC) (R between 0.667 and 0.870) mainly due to the excellent correlation with the inspiratory reserve volume (IRV, R between 0.680 and 0.895) but was poorly correlated with expiratory reserve volume (R below 0.340), RV (R below 0.180), and FRC (R below 0.470). CONCLUSIONS: IS can be used as a simple mean to follow lung function, especially VC and IRV, in the postoperative period in spontaneously breathing patients. IS is noninvasive and can be performed repeatedly at the bedside in the intensive care setting.

Preoperative Predictors of Operative Morbidity and Mortality in COPD Patients Undergoing Bilateral Lung Volume Reduction Surgery—LA Szekely, DA Oelberg, C Wright, DC Johnson, J Wain, B Trotman-Dickenson, J Shepard, DJ Kanarek, D Systrom, LC Ginns. Chest 1997; 111(3):550.

Bilateral volume reduction surgery (VRS) improves lung function for selected patients with emphysema. However, predictors of outcome are not well defined. We reviewed the preoperative characteristics of the first 47 consecutive patients who underwent bilateral VRS at the Massachusetts General Hospital in order to define potential predictors of unacceptable outcome. Preoperative data included spirometry, plethysmography, diffusion of carbon monoxide (D_{CO}), maximum inspiratory pressure (MIP), maximum expiratory pressure, resting arterial blood gases (ABG), cardiopulmonary exercise testing with ABG and lactate sampling, and radionuclide ventriculography. Prepulmonary and postpulmonary rehabilitation 6-minute walk tests (6MWT), and preoperative chest CT scans were also obtained. Twenty-two subjects were male and 17 of the subjects were on the lung transplant list. Patient characteristics included age of 60.5 ± 7.5 years, FEV_1 of 0.67 ± 0.20 L, total lung capacity of 7.56 ± 1.71 L, D_{CO} of 7.40 ± 4.1 mL/min/mm Hg, and P_{aCO_2} of 41.6 ± 6.4 mm Hg (mean ± SD). The FEV_1, vital capacity, MIP, resting room air P_{aCO_2}, prepulmonary and postpulmonary rehabilitation 6MWT, and P_{aCO_2} at maximum oxygen consumption correlated with length of hospitalization ($p < 0.05$). Based on analysis of 41 of 47 patients for whom there were complete data, the inability to walk more than 200 meters on the 6MWT before or after preoperative pulmonary rehabilitation, and resting $P_{aCO_2} \geq 45$ mm Hg were the best predictors of an unacceptable outcome. If either of these characteristics was present, 6 of 16 versus zero of 25 died (Fisher's Exact Test, $p = 0.0025$, one-tailed) and 11 of 16 versus 4 of 25 had hospital courses >21 days ($p < 0.002$). Both the 6MWT < 200 meters and resting $P_{aCO_2} \geq 45$ mm Hg alone correlated with death ($p = 0.004$ and $p = 0.012$, respectively) and the resting $P_{aCO_2} \geq 45$ mm Hg correlated with hospital days >21 ($p = 0.0002$). In conclusion, the data suggest that the inability to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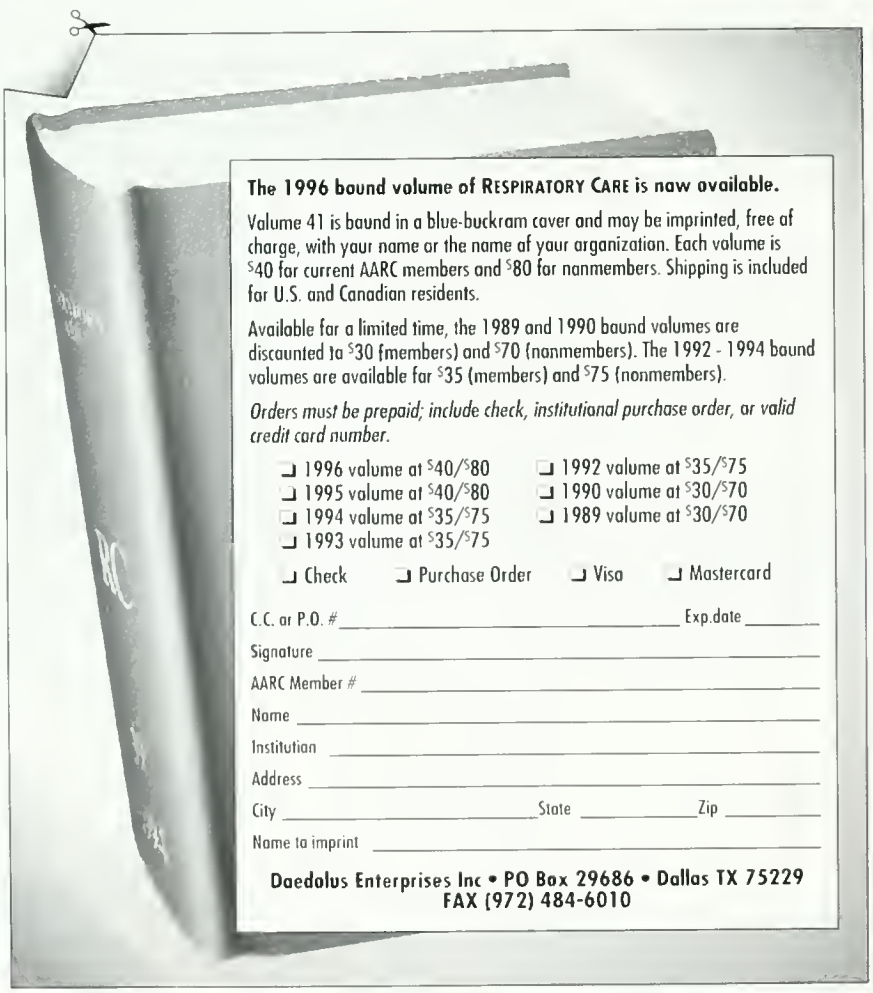
walk at least 200 meters in 6 minutes before or after pulmonary rehabilitation and a resting room air $P_{aCO_2} \geq 45$ mm Hg are excellent preoperative predictors of unacceptable postoperative outcomes.

A Coccidioidomycosis Outbreak Following the Northridge, Calif, Earthquake—E Schneider, RA Hajjeh, RA Spiegel, RW Jibson, EL Harp, GA Marshall, et al. JAMA 1997;277(11):904

OBJECTIVE: To describe a coccidioidomycosis outbreak in Ventura County following the January 1994 earthquake, centered in Northridge, Calif, and to identify factors that increased the risk for acquiring acute coccidioidomycosis infection. DESIGN: Epidemic investigation, population-based skin test survey, and case-control study. SETTING: Ventura County, California. RESULTS: In Ventura County, between January 24 and March 15, 1994, 203 outbreak-associated coccidioidomycosis cases, including 3 fatalities, were identified (attack rate [AR], 30 cases/100,000 population). The majority of cases (56%) and the highest AR (114/100,000 population) occurred in the town of Simi Valley, a community located at the base of a mountain range that experienced numerous landslides associated with the earthquake. Disease onset for cases peaked 2 weeks after the earthquake. The AR was 2.8 times greater for persons 40 years of age and older than for younger persons (relative risk, 2.8; 95% confidence interval [CI], 2.1-3.7; $p < 0.001$). Environmental data indicated that large dust clouds, generated by landslides following the earthquake and strong aftershocks in the Santa Susana Mountains north of Simi Valley, were dispersed into nearby valleys by northeast winds. Simi Valley case-control study data indicated that physically being in a dust cloud (odds ratio, 3.0; 95% CI, 1.6-5.4; $p < 0.001$) and time spent in a dust cloud ($p < 0.001$) significantly increased the risk for being diagnosed with acute coccidioidomycosis. CONCLUSIONS: Both the location and timing of cases strongly suggest that the coccidioidomycosis outbreak in Ventura County was caused when arthrospores were spread in dust clouds generated by the earthquake. This is the first report of a coccidioidomycosis outbreak following an earthquake. Public and physician awareness, especially in endemic areas following similar dust cloud-generating events, may result in prevention and early recognition of acute coccidioidomycosis.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Maternal Smoking and Medical Expenditures for Childhood Respiratory Illness—JJ Stoddard, B Gray. Am J Public Health 1997;87(2):205.

OBJECTIVES: This study sought to assess the association between environmental tobacco smoke exposure from maternal smoking and health care expenditures for respiratory conditions among US children. METHODS: Multivariate analysis of the 1987 National Medical Expenditure Survey was undertaken with a sample that included 2,624 children 5 years of age and under. RESULTS: After analysis that controlled for various sociodemographic factors associated with health care usage, respiratory-related health care expenditures among children whose mothers smoke were found to be significantly higher than those expenditures for children of nonsmoking mothers. Truncated regression techniques were used to estimate that maternal smoking was associated with increased health care expenditures averaging (in 1995 dollars) $120/year for children aged 5 years and under and $175/year for children aged 2 years and under. Our analysis indicates that passive smoking was associated with $661 million in annual medical expenditures in 1987, representing 19% of all expenditures for childhood respiratory conditions. CONCLUSIONS. Maternal smoking is associated with significantly increased child health expenditures and contributes significantly to the overall cost of medical care.

Inhaled Steroids and the Risk of Hospitalization for Asthma—JG Donahue, ST Weiss, JM Livingston, MA Goetsch, DK Greineder, R Platt. JAMA 1997;277(11):887.

OBJECTIVE: To determine if anti-inflammatory treatment for asthma reduces the risk of asthma hospitalization. DESIGN: Retrospective cohort study. SETTING: A health maintenance organization (HMO) in eastern Massachusetts. PARTICIPANTS: Members of the HMO who were identified during the period October 1991 through September 1994 as having a diagnosis of asthma using a computerized medical record system. MAIN OUTCOME: Hospitalization for asthma. RESULTS: Of the 16,941 eligible persons, 742 (4.4%) were hospitalized for asthma. The overall relative risk (RR) of hospitalization among those who received inhaled steroids was 0.5 (95% confidence interval [CI], 0.4-0.6) after adjustment for β-agonist dispensing. Additional adjustment for age, race, other asthma medications, and amount and type of ambulatory care for asthma did not substantially affect the inverse relationship between use of inhaled steroids and hospitalization. Cromolyn was similarly associated with reduced risk, especially among children (RR, 0.8; 95% CI, 0.7-0.9). In contrast, increasing β-agonist use was associated with increasing hospitalization risk even after adjustment for other factors and medications. The steroid-associated protection was most marked among individuals who received the largest

amount of β-agonist. CONCLUSIONS: Inhaled steroids and, to a lesser extent, cromolyn confer significant protection against exacerbations of asthma leading to hospitalization. These results support the use of inhaled steroids by individuals who require more than occasional β-agonist use to control asthma symptoms.

The Use of Verbal Expectancy in Reducing Pain Associated with Arteriotomies—F Austan, M Polise, TR Schultz. Amer J Clin Hypn 1997;39(3):182.

Hospitalized patients (n = 25) receiving arteriotomies were given 1 of 2 verbal instructions 1 hour apart prior to each incision. In the first arteriotomy, the right radial artery was prepped with alcohol without mention and administered non-expectancy instruction-A, "You may or may not feel pain, everyone is different." One hour later, prior to the second incision, prepping the left radial artery with alcohol was brought to the patient's attention while providing expectancy instruction-B, "Notice how cool this feels, it' s interesting how coldness numbs the skin." Analysis of variance revealed that administration of expectancy instruction-B significantly reduced pain ($p < 0.005$, determined by paired *t*-test) associated with arteriotomies.

Tubefeeding and Mortality in Children with Severe Disabilities and Mental Retardation—D Strauss, T Kastner, S Ashwal, J White. Pediatrics 1997;99(3):358.

OBJECTIVE: To study the contribution of tubefeeding to mortality for children with severe disabilities and mental retardation. Previous research has suggested an association between tubefeeding and mortality. However, risk has never been determined using population-based data or defined in regard to patient variables. METHODS: Retrospective analysis of a comprehensive statewide data set comprised of 4,921 children with severe disabilities and mental retardation living in community and congregate care settings. The outcome measure was mortality; primary study variables included the presence of a feeding tube, measures of functional independence, type of residence, and medical comorbidity. RESULTS: There were 4 findings. First, the use of a feeding tube was associated with virtually every disability. Second, when no study variables were controlled, statistically significant differences in mortality rates were noted between children who were tubefed and those who were not. The relative risk of mortality associated with use of a feeding tube was 2.1. Third, the use of a feeding tube was associated with a reduction in relative risk of mortality in children with tracheostomy (relative risk of mortality: 0.55). However, this association did not achieve statistical significance. Fourth, when study variables were controlled in a multivariate analysis, feeding tube use was associated with no identifiable increase in mortality among children with very severe disabilities, but was associated with an approximated doubled mortality rate among those with less severe disabilities. CONCLUSIONS: We hypothesize that the increased mortality associated with tubefeeding may be attributable to a differential increase in pulmonary disease secondary to overly vigorous nutritional maintenance and subsequent aspiration after tube placement. For children with tracheostomy this risk may be reduced. If tracheostomy proves to be associated with a relatively more favorable outcome for tubefeeding, we hypothesize that it would reflect the benefits of tracheostomy in allowing access to the airway for suctioning and ventilation. Given the observed higher mortality rates among the less severely disabled children who are tubefed and the substantial costs associated with tubefeeding, a prospective, controlled study may be clinically indicated, ethically justifiable, and economically warranted.

Risk Status at Discharge and Cause of Death for Postneonatal Infant Deaths: A Total Population Study—A Kempe, PH Wise, NS Wampler, FS Cole, H Wallace, C Dickinson, et al. Pediatrics 1997;99(3):338.

OBJECTIVES: To obtain population-based, clinical information regarding potentially modifiable factors contributing to death during the postneonatal period (28 to 364 days), we examined all postneonatal infant deaths in 4 areas of the United States to determine: (1) the cause of death from clinical and autopsy data rather than vital statistics, (2) whether death occurred during initial hospitalization or after discharge, and (3) the portion of postneonatal mortality attributable to infants who left the hospital with identified high-risk medical conditions. DESIGN & SETTING: Retrospective medical record review of all postneonatal infant deaths with birthweights greater than 500 g (total n = 386) born to mothers residing in: (1) the city of Boston (1984 and 1985, n = 55), (2) the city of St Louis and contiguous areas (1985 and 1986, n = 123), (3) San Diego County (1985, n = 112), and (4) the state of Maine (1984 and 1985, n = 96). Deaths were identified using linked birth and death vital statistics, and medical record audits of infants' and mothers' charts were performed. Causes of death were obtained from medical record review in conjunction with autopsy if performed (72%, n = 278), medical record alone (17%, n = 67), or vital statistics if no other source was available (11%, n = 41). The medical conditions at the time of discharge for each infant were reviewed and, if judged to confer an increased risk of morbidity or mortality, were classified as high risk. RESULTS: The causes of death were sudden infant death syndrome (47%, n = 181), congenital conditions (20%, n = 77), prematurity-related conditions (11%, n = 43), infections (9%, n = 34), external causes (including injuries, drownings, ingestions, and burns) (7%, n = 25), and other (6%, n = 23). In 24% of congenital and 25% to 44% of prematurity-related deaths, infection was the acute or associated cause of death. Infants born to black mothers were more likely than those born to white mothers to die during the postneonatal period of all major causes of death (7.3/1,000 vs 3.0/1,000). Overall, 18% (n = 68) of deaths occurred to infants who never left the hospital; 79% (n = 305) of the infants were discharged before death; and discharge status was unknown in 3% (n = 13). Eighty-one percent of all infants with prematurity-related postneonatal deaths were never discharged, and of the total infants who were initially discharged, only 1% (n = 4) subsequently died of prematurity-related causes. Of all postneonatal deaths, only 16% (n = 62) left the hospital with identified high-risk medical conditions. CONCLUSIONS: These findings suggest that the etiology of postneonatal mortality is heterogeneous, with significant complexity in attributing specific causes of death and making designations of "preventability." The vast majority of infants who died of prematurity-related postneonatal causes never left the hospital, and only a small percentage of all infants that left the hospital before death were identified as being at high medical risk. Therefore, strategies for further decreasing postneonatal mortality must link high-risk follow-up programs to more comprehensive strategies that address risk throughout pregnancy and early childhood.

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10:00 a.m. - 10:05 a.m.
Program Overview
Patrick J Dunne MEd RRT, Southwest Medical Emporium, Fullerton CA

10:05 a.m. - 10:50 a.m.
The Needs of Health Care Systems in the Future—Emerging Trends and Expanding Skills Inventory
Patrick J Dunne MEd RRT
Presents the forces impacting on health care delivery and the response by delivery systems. Reviews past, present, and future reimbursement mechanisms and the response of delivery systems to incentives they create. Explains the impact of expanding post acute services, the opportunities for RCPs, and the skill sets required to add value in future health care systems.

10:55 a.m. - 11:40 a.m.
Providing Value in Acute Care Facilities—Managing Demand for Services
Kevin L Shrake MA RRT FACHE, Memorial Medical Center, Springfield IL
Defines the value added by RCPs in present and future acute care facilities. Describes how to sell decision makers on the value of using guidelines and protocols to assure appropriate utilization. Defines resources and support required to implement a utilization control program. Presents a case study of a department that has been successful in assuring appropriate utilization.

11:45 a.m. - 12:30 p.m.
The Post Acute Environment—Skilled Nursing Facilities, Subacute, and Home Care
Dianne L Lewis MS RRT, Acute and Subacute Care and Management Consultant, Naples FL
Identifies market forces and government regulations that foster growth in the post acute environment. Discusses the opportunities for RCPs created by government and private managed care contracting. Reviews the mechanism for reimbursement of respiratory care services in post acute sites and the current HCFA scrutiny of transfer agreements.

SUCCESSFUL STRATEGIES FOR TOMORROW'S RESPIRATORY CARE SERVICES

1:30 p.m. - 2:25 p.m.
Respiratory Care in Group Practice and Disease Management
Patrick J Dunne MEd RRT
Explores opportunities for RCPs as mid-level providers in capitated physician group practice. Defines disease management and explains why asthma patients are ideal candidates for disease management programs. Identifies skill sets required by mid-level providers and explains why RCPs add unique value to respiratory disease management programs.

2:30 p.m. - 3:25 p.m.
Respiratory Care in the Future: Integrated Care Delivery Systems
Dianne L Lewis MS RRT
Describes market forces encouraging the development of integrated care delivery systems. Reviews current and projected penetration of managed care across the nation. Explains the components of an integrated delivery system and the competitive advantage afforded by the system. Presents a proposed respiratory care service structure to meet the needs of an integrated care delivery system.

3:30 p.m. - 4:25 p.m.
Marketing and Selling Your Service and Your Services
Kevin L Shrake MA RRT FACHE
Presents a systematic approach to developing new or expanded services. Discusses the fundamentals of a business plan and techniques for successfully presenting ideas to organizational decision makers. Reviews common pitfalls to avoid.

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Nose Clips: Why Worry about Them?

In this issue of the Journal, you will find our study evaluating the use of nose clips (NC) during performance of the standard forced expiratory maneuver (spirometry). Why did we spend the time and energy to address this issue? Is it really worthwhile or necessary to determine whether NC are beneficial during spirometry? After all, NC have been used for decades with good results, are inexpensive, easy to apply, and well tolerated. On the other hand, NC have to be purchased, cleaned, reused, and eventually discarded to find their way to landfills. If, for some reason, none are available at the time a patient is ready to perform spirometry, then one has to go searching for them. Some patients, especially children, may be distracted by them, and time might have to be taken to convince a patient to apply them.

Although both the American Thoracic Society[1] and the European Respiratory Society[2] consensus statements favor the use of NC during spirometry, neither references studies that support this. We performed a literature review and could find no studies that demonstrated a benefit from NC use during spirometry. However, we did find 2 investigations that found no difference in spirometric values with NC.[3,4]

SEE THE RELATED EDITORIAL ON PAGE 489 AND THE ORIGINAL STUDY ON PAGE 492.

To determine how directors of pulmonary function laboratories feel about the use of NC, we mailed a questionnaire to the directors of 76 pediatric and 100 adult pulmonary function laboratories across the country. We asked 3 questions: (1) How often do you use NC during standard forced expiratory spirometry? (2) If you use NC regularly, why? (3) If a well-designed study demonstrated that NC made no difference, would you consider discontinuing use? We chose the pediatric pulmonary function laboratories from the 1993 listing of Cystic Fibrosis Centers and sent a questionnaire to one or more centers from each State. The adult laboratories were chosen from the 1994 American Hospital Association Guide. Two hospitals identified as having pulmonary function laboratories, 1 with < 200 beds and 1 with > 200 beds, were chosen randomly from each of the 50 states. We received responses from 66 (86%) of the pediatric and 64 (64%) of the adult laboratories. The responses from the pediatric and adult laboratories were similar. Approximately 90% of the laboratories reported using NC routinely during the standard forced expiratory maneuver. The main reasons given were that they were recommended by the ATS and that laboratory directors believed that use of NC resulted in better spirometric values. Of the laboratories that used NC routinely, 36 (61%) of the pediatric and 34 (57%) of the adult laboratory directors replied that they would consider discontinuing the routine use of NC if a well-designed study demonstrated that they were not beneficial. However, approximately 40% stated that they would continue to use NC despite such a study.

It is interesting that NC use is so widely implemented for forced expiratory spirometry considering the lack of scientific evidence that supports their use. In addition, many of us seem unwilling to forego them even if new data show that they are not necessary.

Why are we so unwilling to relinquish NC? Is it because they really work or because we just think they do? Despite the paucity of data supporting their use, the responses to our questionnaire suggest that many laboratories will be unwilling to stop using NC even if data are published demonstrating that they are not necessary. Do these laboratories have data obtained in a rigorous manner that support their use? If they do, they should submit those data for publication. More likely the resistance to change is due to an unwillingness to alter something that works.

The paradigm is that NC should be used when we perform spirometry, and we are reluctant to reject paradigms, especially if continuing them results in no harm. Francis Bacon, the 17th Century philosopher, stated "It is true that what is settled by custom, though it not be good, yet at least it is fit."[5] It is our 'custom' to use NC, but we should be willing to reconsider use if new data support a change in the paradigm. Thomas Carlyle, the 19th Century British thinker, wrote "Custom makes dotards of us all. ...Philosophy complains that Custom has hoodwinked us, from the first; that we do everything by Custom, even Believe by it; that our Axioms, let us boast of Free-thinking as we may, are oftenest simply such Beliefs as we have never heard questioned."[6] Whether it be using NC for

spirometry or anything else that we do in medicine, we should be able to objectively assess the validity of our actions based upon the scientific evidence. Why fuss about NC? Simply because we should not be satisfied with doing something merely because it is 'custom' but rather because critical evaluation of the data supports it.

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Not Using Nose Clips: Another Thing To Worry About?

In this month's Journal, Pina et al[1] present the results of a study designed to answer the question, Are nose clips necessary when performing forced expiratory spirometry? The authors executed a well-designed, single-masked, randomized crossover study that compared the FVC, FEV_1, $FEF_{25-75\%}$, and PEF obtained with and without nose clips, in a group of patients with and without airflow obstruction. The authors conclude that nose clips are not routinely necessary when performing spirometry.

The results were not surprising to me, I have occasionally forgotten to replace nose clips between a patient's spirometry efforts, yet, have seen excellent agreement between results obtained with and without nose clips. After reading the paper, I intentionally performed some tests without nose clips in the interest of finding whether the conclusion of this study could be applied by practitioners who utilize a modified technique[2] for performing spirometry. My anecdotal data support the notion that the results will be equivalent even when the modified spirometry technique is used.

SEE THE RELATED EDITORIAL ON PAGE 487 AND THE ORIGINAL STUDY ON PAGE 492.

However, I am not likely to change my position that nose clips should be used routinely during spirometry. Although it is comforting to know that the data is likely to be just as good without nose clips, I take even greater comfort in using nose clips, knowing that there is one less thing to worry about during testing. I view each spirometry as a challenge to obtain the highest quality data possible. Nose clips provide assurance that I am dealing with a truly closed system; this allows me to focus my attention on the patient and the test data.

The authors correctly point out that the American Thoracic Society (ATS) Spirometry Standardization Statements[3-5] provide no references to support the recommendation that nose clips be used. However, personal communications, between members of the ATS Committees crafting the Statements and members of the Clinical Practice Guideline Focus Group that prepared the first Guideline[6] and the 1996 Update,[7] suggest that the recommendation came only after a great deal of thought and discussion. (Personal communication from Robert Crapo MD and Charles Irvin PhD to the Focus Group 1991 and to Jack Wanger 1996; Reed Gardner PhD to Pat Brougher 1987.)

I am concerned that the conclusions of the authors may be shortened to the general rule, "nose clips are not necessary when performing spirometry." Omission of the word, routinely, while innocent at first blush, changes the flavor of the authors' conclusion in an important way. The authors clearly state that there are circumstances in which nose clips should be used, and they wisely suggest methods for determining when this is appropriate.

All practitioners who opt to omit the use of nose clips as part of their testing routine must remember this additional potential source of error. The authors suggest that the loss of air through the nose when nose clips are not used is likely to be rare, but this may be sufficient reason to recommend their continued use. If the occurrence is infrequent, the problem may not be noticed. Of particular concern is the situation in which the individual administering the test is relatively inexperienced or administers the test infrequently. In the end, readers must decide for themselves if they wish to adopt a practice that carries the price tag of additional vigilance.

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The Battle for Redesign: Time for a New Strategy?

> Let us cross over the river and rest in the shade of the trees.
> *—the dying words of General "Stonewall" Jackson*

While to say that the world of modern health care is in a state of war may be somewhat extreme, that world is certainly engaged in a struggle of unknown proportions. Our profession is working overtime to survive and—we hope—to flourish. We must remember that all members of the health care team are doing the same. Everywhere we look we see physicians, nurses, and allied health care professionals contending to maintain their positions within our rapidly changing health care environment. Maybe it's time to stop and look at the situation carefully before continuing to work ourselves into oblivion.

As we approach the next millennium, most of us are working to develop therapist-driven protocols (TDPs), respiratory consult services, care maps, clinical practice guidelines (CPGs), critical pathways, benchmarking—and on and on it goes. In hospitals across the country, respiratory care managers are attending committee meetings and training seminars from sunup to sundown. Although it appears that in most cases our efforts are well spent, it may be time to regroup. It is fortunate that the current climate has caused us to pause and take a serious look at our profession. After all, when threatened, we either stay and "fight" or take "flight." Some have flown, others have stayed to fight. It's time we review our battle plan.

Sam Giordano has identified one aspect of the struggle that needs careful consideration: our relationship with physicians. In fact, he says that it is "the key to our future."[1] All of our current efforts—TDPs, CPGs, and all—have a common vital component: the physician! If one carefully reads the Conference Proceedings of Emerging Health Care Delivery Models and Respiratory Care in the January 1997 issue of RESPIRATORY CARE one notes the importance of physicians, and more importantly our relationship to them. I am not so naive as to think that physicians are not going through some drastic changes of their own. In fact, as we experience some frustration in our own profession, just stop and think about the level of frustration many physicians are experiencing as they now answer to managed care CEOs who may be far removed from the front line (ie, the bedside). However, they are the generals on the front line; and, regardless of how the health care terrain is redesigned, I believe that the physician will invariably maintain that position! However, we and many physicians are not on the same battlefield. We have to remind ourselves that we are in the same war: the effective and efficient delivery of quality health care. I don't believe that, in some hospitals, our generals are going to the same meetings and struggling with the same questions that we are as we grapple to redesign the way we deliver health care. How can we address this issue in our individual departments and as a profession?

First, respiratory care practitioners need to communicate with physicians as fellow scientists. It may be that many physicians don't perceive us as fellow scientists because we have been so busy performing tasks. We all seem to recognize that our relationship with the physician is paramount; however, the importance of recognizing and nurturing that relationship was not explicitly or implicitly conveyed in a description of the attributes and characteristics needed by future respiratory care practitioners.[2] It is interesting that in another recent consensus of respiratory care educators, "research" was specified as being within the future scope for professional respiratory care practitioners.[3] This is a move in the right direction! However, most of the respiratory care students I work with see no connection between a student research project and their future in our profession! It may be time to stop and go rest under the shade of the trees and learn how to relate to our fellow soldier, the physician!

Some of our time may best be spent in the library and not in running from patient to patient giving nebulizer treatments. I know this sounds impossible for those of us who are not in large research and teaching institutions. Of the 132 abstracts presented in the 1996 OPEN FORUM and published in the October issue of RESPIRATORY CARE, only 3 were submitted from hospitals not affiliated with medical schools! It is obvious that most research is done by our colleagues in the larger teaching hospitals, but it doesn't have to remain that way!

Due to the foresight of a dedicated and insightful respiratory care manager, who sent me to a research and writing workshop at a national AARC convention, I have been allowed to perform, and fortunately publish, three articles (a fourth is in press, a fifth is in review) in the last couple of years. I have been able to involve several key physicians within our midsize, not-for-profit, community hospital. I believe this is a highly fruitful way to strengthen relationships with physi-

cians within our institutions.[4] This is time well spent! Our department has enjoyed the fruits of what we can call image transformation efforts. It is true that free pizza at department meetings will make a friend for a while, but co-authoring a case study in a recognized medical journal will make a colleague for a lifetime. More importantly, many of us now communicate with physicians as fellow scientists because they now understand that we are their fellow scientists! The benefits of getting published may have a far-reaching impact, making the ulcers, gray hair, and insomnia seem worth it.

Second, we need to learn from our mistakes. If our partnership with physicians is fundamental, and I believe that it is, then we need to involve them more in the major undertakings of our profession. Our profession is blessed with many outstanding physician advocates; however, we need more physicians in the trenches to understand and value our profession. One major project I am referring to is the development of the CPGs. Although physicians have worked with CPG Focus Groups and served as reviewers, we need to recognize that more formal review and approval by physician groups like the American Thoracic Society or American Society of Anesthesiologists may be an astute move in the overall battle.[5] (Only 18 of the current 46 CPGs in publication had physician representation on their individual guideline committees.) Sometimes eating crow may be the best diet for the sake of the profession.

In closing, I ask you as my fellow professionals to consider developing a closer working relationship with the physicians within your institution. They are your friends not your foes. Don't get hit by 'friendly fire' due to the lack of a genuine professional relationship. Do they look at you as a fellow scientist? Can you relate to them as a fellow scientist? Would you be better off spending time reading *Chest*, RESPIRATORY CARE, or *Critical Care Medicine*, than the latest account of health care management techniques that may promise more than they deliver? Lastly and respectfully, should we look back and say that we need to include more physicians in the defining and developing process of our profession? Let us stop and rest on these questions.

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Are Nose Clips Necessary in Adults and Children Performing Forced Expiratory Spirometry?

Major Joseph S Pina MC MD, Captain Melinda L Behrens MC MD, Colonel Donald R Moffitt MC MD, Major (P) Bernard J Roth MC MD, and Lieutenant Colonel Edward R Carter MC MD

BACKGROUND: Although the use of nose clips (NC) during forced expiratory spirometry is encouraged by both the American Thoracic Society (ATS) and the European Respiratory Society (ERS), few data support this practice. METHOD & SUBJECTS: We performed a prospective, single-masked, crossover study in children and adults to determine whether the use of NC significantly affected spirometric values. Eighty adults (46% with airways obstruction) and 81 children (22% with airways obstruction) performed spirometry both with and without (w/o) NC in random order. RESULTS: For children, no significant differences were found between spirometric values (FVC, FEV_1, $FEF_{25-75\%}$, and PEFR) obtained with and w/o NC. For adults, there were no significant differences in FVC or PEFR. The difference in FEV_1, although only 1% of the mean value, was statistically significant; the mean (SD) for FEV_1-NC was 3.03 (1.14) L versus 3.00 (1.12) L w/o NC, p = 0.001. No children and 1 adult had a > 10% improvement in FEV_1 with NC, and 2 patients (both children) had a > 10% improvement w/o NC. The mean differences were similar and not statistically significantly different for obstructed and unobstructed patients. We found that the use of NC did not alter values obtained by forced expiratory spirometry in a clinically important manner. CONCLUSION: Our results suggest that the routine use of NC during forced expiratory spirometry should be questioned. [Respir Care 1997;42(5):492-497]

Background

The American Thoracic Society (ATS)[1] and the European Respiratory Society (ERS)[2] recommend the use of nose clips (NC) when performing forced expiratory spirometry. In theory, NC prevent air from escaping through the nose, but there is little evidence, if any, that significant volume is lost during a forced expiratory maneuver in a cooperative patient. In fact, data suggest that the posterior soft palate moves cephalad during the maneuver completely interrupting nasal air flow.[3] In addition, Verral and colleagues[4] found no significant differences between spirometric values obtained with and without (w/o) NC in a group of healthy adult men.[4] Thus, the use of NC during forced expiratory spirometry may be unnecessary.

There are several potential reasons to consider not using NC. Some patients, especially children, may find the wearing of NC uncomfortable or distracting, and young children may even fight the placing of NC. Even though NC are well tolerated by the majority of patients, it may be more convenient

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not to use them. Therefore, to determine whether the use of NC significantly affected values obtained during forced expiratory spirometry we performed a randomized, cross-over, investigator-masked study in children and adults who presented to the pulmonary function laboratory for testing. The primary outcome measure was the difference in the values for FEV_1 (volume exhaled in the first second) obtained with and w/o NC.

SEE THE RELATED EDITORIALS ON PAGE 487 AND PAGE 489.

Method & Subjects

Study Population

Children 6-18 years old who presented to the Madigan Army Medical Center Pediatric or the Mary Bridge Children's Hospital Cystic Fibrosis Center pulmonary function laboratories and adult patients presenting to the Tripler Army Medical Center pulmonary function laboratory were eligible for the study. Patients were excluded from enrollment if they were unable to perform spirometry properly. For children we defined airways obstruction as an $FEV_1 < 80\%$ of their best ever value and the ratio of FEV_1 to forced vital capacity (FEV_1/FVC) < 0.8. For adults we used an FEV_1 and an FEV_1/FVC that were both lower than the 95% confidence intervals as determined by Morris and co-workers.[5] The children were subdivided into 4 groups; Group 1 is ≤ 10 years old who had never performed spirometry before, Group 2 is ≤ 10 years old who had performed spirometry before, Group 3 is > 10 years old who had never performed spirometry before, and Group 4 is > 10 years old who had performed spirometry before.

Study Design

This was a prospective, randomized, cross-over, investigator-masked study. The protocol was approved by the Madigan Army Medical Center and Tripler Army Medical Center Institutional Review Boards. Informed written consent was obtained from all subjects and, for the children, from their parents or legal guardians as well. Once enrolled, subjects performed spirometry with and w/o NC in random order as determined by withdrawing from an envelope a slip that directed the patient to use NC or no NC first. Patients performed a minimum of 3 forced expiratory maneuvers both with and w/o NC and rested for 1-5 minutes between the series of maneuvers. Values were saved using ATS criteria,[4] and trials that were not acceptable by these criteria were rejected. If necessary, subjects performed additional trials (up to 6) until 3 trials had FVCs that were within 5% of each other. If a trend towards improvement was noted in consecutive trials within a session, then further trials were performed until 3 values with FVCs within 5% of the best trial were observed. From the acceptable curves in each session, we then saved the largest FVC, FEV_1, and PEFR (even if the values came from different trials), and the $FEF_{25-75\%}$ from the trial with the largest FVC + FEV_1. The investigator or technician was masked to the use of NC by a curtain separating her/him from the patient. An assistant applied the NC at the appropriate time and observed the patient during the maneuver to help ensure that the patient was performing the test correctly. However, the assistant was not allowed to coach the patient during the maneuver.

Subjects were withdrawn from the study if (1) they could not perform the forced expiratory maneuver consistently throughout the study period, (2) they demonstrated a progressive drop in FEV_1 of ≥ 10% with successive maneuvers (indicating bronchospasm induced by performing the maneuver), or (3) they were noted to deteriorate clinically.

Measurements

At the MaryBridge Cystic Fibrosis Clinic, FVC, FEV_1, FEF_{25-75}, and PEFR were measured with a Cybermedic spirometric system (Cybermedic, Boulder CO). At the Madigan Army Medical Center, measurements were obtained with a rolling seal spirometer (Collins Co, Braintree MA), and at the Tripler Army Medical Center a Sensormedics Vmax 229 spirometer (Sensormedics Corporation, Yorba Linda CA) was used. Spirometric values for the children and adults were expressed in absolute values. The FVC and FEV_1 were expressed in liters and the PEFR and FEF_{25-75} in L/sec.

Statistical Analysis

The primary outcome variables were the means of the individual differences between spirometric values obtained with and w/o NC, especially FEV_1. Before beginning the study we performed a sample size determination. Accepting an α of 0.05, α-β of 0.1, and a standard deviation (SD) of 225 mL (estimated from an expected range of individual differences in FEV_1 of −450 to +450 mL = 900 mL divided by 4), we estimated that we would need a minimum of 25 children and 25 adults to detect a mean difference in FEV_1 of 150 mL. (For an FEV_1 of 3 L, this would be a 5% change.) The Student's *t* test for repeated measures was used to analyze intra-group differences between spirometric values obtained with and w/o NC. A p value of < 0.05 was considered significant, and no correction for multiple comparisons was used. One-way ANOVA was used to analyze intergroup differences in spirometric values obtained with and w/o NC. All data are presented as mean (SD).

A limits-of-agreement analysis was performed on both the combined pediatric and the combined adult data according

to the method outlined by Bland and Altman.[6] This analysis consisted of plotting the difference between the spirometric values for each patient (value obtained with NC minus the value obtained w/o NC) against the average value for that patient ([value obtained with NC + the value obtained w/o NC]/2). The mean of these individual differences ± 2 SDs provided the limits of agreement with approximately 95% of all the individual differences falling within this range.

Results

Eighty-one children [51 boys, 30 girls, mean age 10.9 (3) y] and 89 adults [48 men, 32 women, mean age 44 (17) y] were enrolled in the study. The children were enrolled from August 1994 through June 1995, and the adults were enrolled from October through December 1996. Of the adults entered, 9 had to be withdrawn from the study (6 because they could not perform spirometry consistently and 3 due to worsening bronchospasm). No children were withdrawn. There were 12 children in Group 1, 26 in Group 2, 12 in Group 3, and 30 in Group 4. Of the pediatric subjects, 59 had asthma and 12 had cystic fibrosis. Of the adults, 15 were diagnosed with chronic obstructive pulmonary disease and 22 with asthma. Forty-six percent of the adult subjects (37/80) and 22% (18/81) of the pediatric subjects met the criteria for airways obstruction.

All patients tolerated NC well. The children were not specifically asked their preference concerning the use of NC, but 39% of adult patients stated that they would rather not use NC. 48% had no preference, and 14% preferred using NC. Only 1 adult and none of the children exhibited obvious nasal breathing at the onset of testing. In the 1 adult, breathing was audible, and the FVCs were grossly inconsistent. This patient corrected his technique immediately with further instruction.

The means of the individual differences in spirometric values obtained with and w/o NC did not differ significantly among any of the patient subgroups as determined by one-way ANOVA. For the children, no significant differences were observed among values obtained with and w/o NC no matter how the children were subdivided. Similarly, there were no significant differences in values obtained with and w/o NC between the group of adults with airways obstruction and the group w/o obstruction.

The means of the individual differences in spirometric values obtained with and w/o NC for the various groups of patients are presented in Table 1. For the children, no statistically significant differences in any of the spirometric values were observed with and w/o NC. For adults there were no statistically significant differences in FVC or PEFR, but the difference in FEV_1 did reach statistical significance in favor of NC for the unobstructed patients and the total group but not for the obstructed group. The difference in FEV_1 for the total group of adults was 0.03 L, which was only 1% of the averaged values for FEV_1—3.03 L (NC) and 3.00 L (w/o NC). The difference in FEF_{25-75} was also noted to be statistically significant for the total group of adults but not for the obstructed and unobstructed groups when analyzed separately.

Table 1. Spirometric Values [Mean (SD)] Obtained With (NC) and Without Nose Clips (w/o NC)

Patient Group	FVC			FEV_1			PEF			FEF_{25-75}		
	NC (L)	w/o NC (L)	p Value*	NC (L)	w/o NC (L)	p Value	NC (L/s)	w/o NC (L/s)	p Value	NC (L/s)	w/o NC (L/s)	p Value
Adults												
Obstructed (n = 37)	3.69 (1.10)	3.71 (1.12)	0.49	2.34 (0.78)	2.31 (0.78)	0.16	6.47 (2.29)	6.38 (2.20)	0.39	1.42 (0.87)	1.38 (0.89)	0.15
Unobstructed (n = 43)	4.43 (1.22)	4.42 (1.21)	0.39	3.63 (1.05)	3.60 (1.03)	0.03	9.10 (2.51)	8.98 (2.53)	0.10	3.79 (1.34)	3.72 (1.33)	0.14
Total (n = 80)	4.09 (1.22)	4.09 (1.14)	0.93	3.03 (1.14)	3.00 (1.12)	0.001	7.89 (2.73)	7.78 (2.70)	0.08	2.69 (1.65)	2.63 (1.64)	0.05
Children												
Obstructed (n = 20)	2.47 (0.87)	2.50 (0.90)	0.38	1.69 (0.56)	1.71 (0.54)	0.08	4.40 (1.23)	4.43 (1.23)	0.62	1.20 (0.43)	1.17 (0.41)	0.25
Unobstructed (n = 61)	2.50 (0.91)	2.49 (0.93)	0.42	2.13 (0.73)	2.13 (0.75)	0.75	4.85 (1.48)	4.88 (1.56)	0.62	2.40 (0.88)	2.36 (0.91)	0.13
Total (n = 81)	2.50 (0.89)	2.50 (0.91)	0.94	2.02 (0.71)	2.03 (0.73)	0.68	4.74 (1.43)	4.77 (1.49)	0.51	2.10 (0.95)	2.06 (0.96)	0.07

*p values for the intra-group differences by student's *t* test for paired data. No statistically significant differences in spirometric values were obtained with and w/o NC between any of the groups (ie, obstructed vs unobstructed, or adults vs children) as determined by one-way ANOVA.

A limits-of-agreement analysis was performed on the total group of children and also for the total group of adults for each of the spirometric measures (Figures 1 and 2). All of the mean differences were near zero, and approximately 95% of the individual differences fell within ± 0.25 L for FVC, 0.16 L for FEV_1, 1.0 L/sec for PEFR, and 0.60 L/sec for FEF_{25-75}.

To determine how many patients had clinically important differences in spirometric values either with or w/o NC, we determined the number of patients who had a >10% improvement in FVC, FEV_1, and PEFR with NC and also the number of patients who had a >10% improvement w/o NC (Table 2). We performed the same calculations for FEF_{25-75} using a >20% improvement. Of the total patient group of 81 children and 80 adults, 1 patient had a >10% improvement in FEV_1 with NC, and 2 patients had a >10% improvement w/o NC.

Table 2. Number of Patients with Clinically Important Improvements in Spirometric Values, with Nose Clips (NC) or without Nose Clips

	> 10%* in FVC	> 10% in FEV_1	> 10% in PEFR	> 20% in FEF_{25-75}
Adults				
Using NC	4	1	6	6
Not using NC	7	1	3	1
Children				
Using NC	1	0	6	4
Not using NC	1	1	9	2

*The % improvement with NC = [value with NC – value w/o NC]/value with NC × 100. The % improvement without NC = [value w/o NC – value with NC]/value w/o NC × 100.

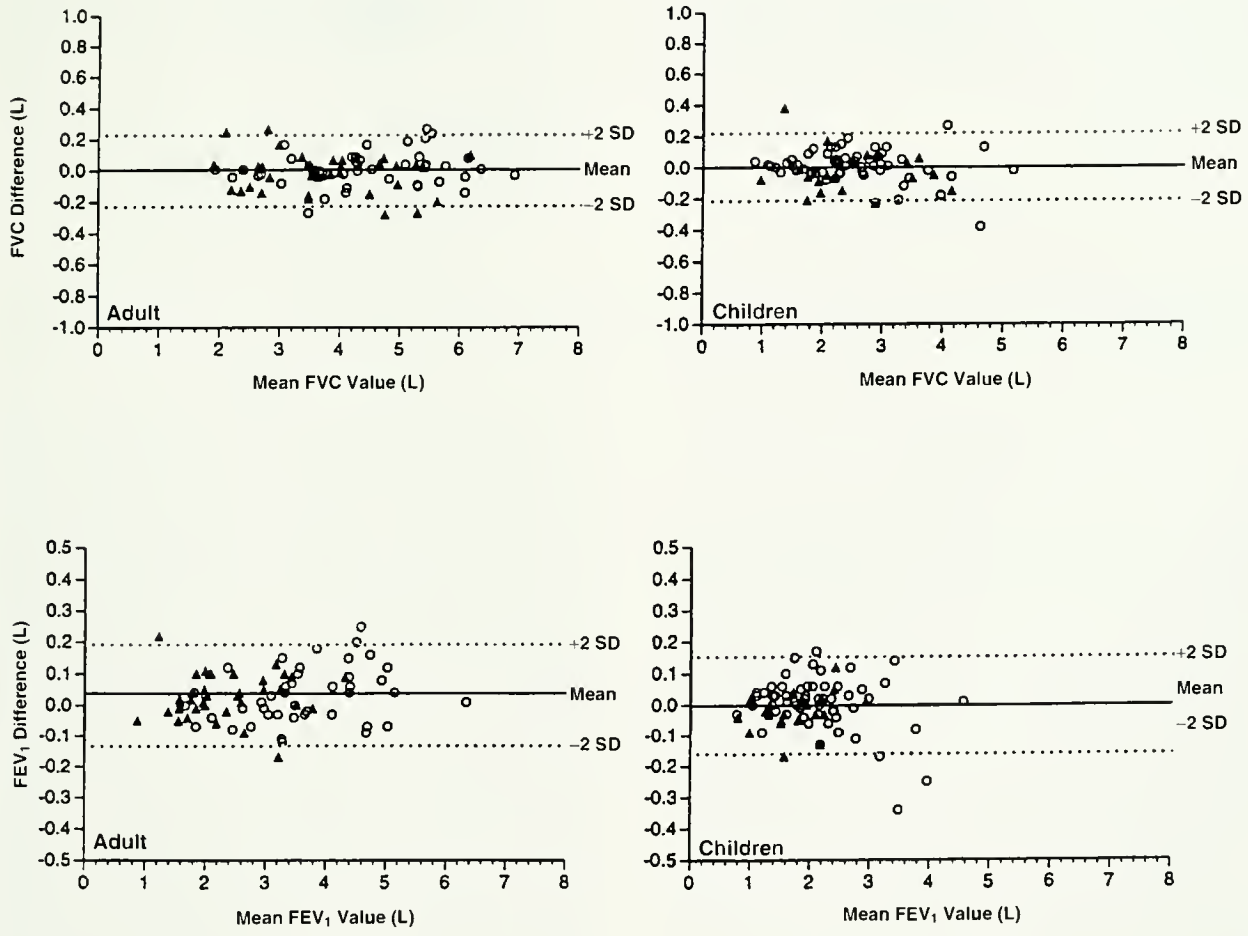

Fig. 1. Each individual difference in FVC and FEV_1 (value obtained with NC – value obtained without NC) is plotted against the mean value for that patient ([value obtained with NC + value obtained without NC]/2). Δ = patients with airways obstruction (see text for criteria), and o = subjects without airways obstruction. The solid line designates the mean of the individual differences for the group, and the dotted lines designate ± 2 SD, ie, the limits of agreement.

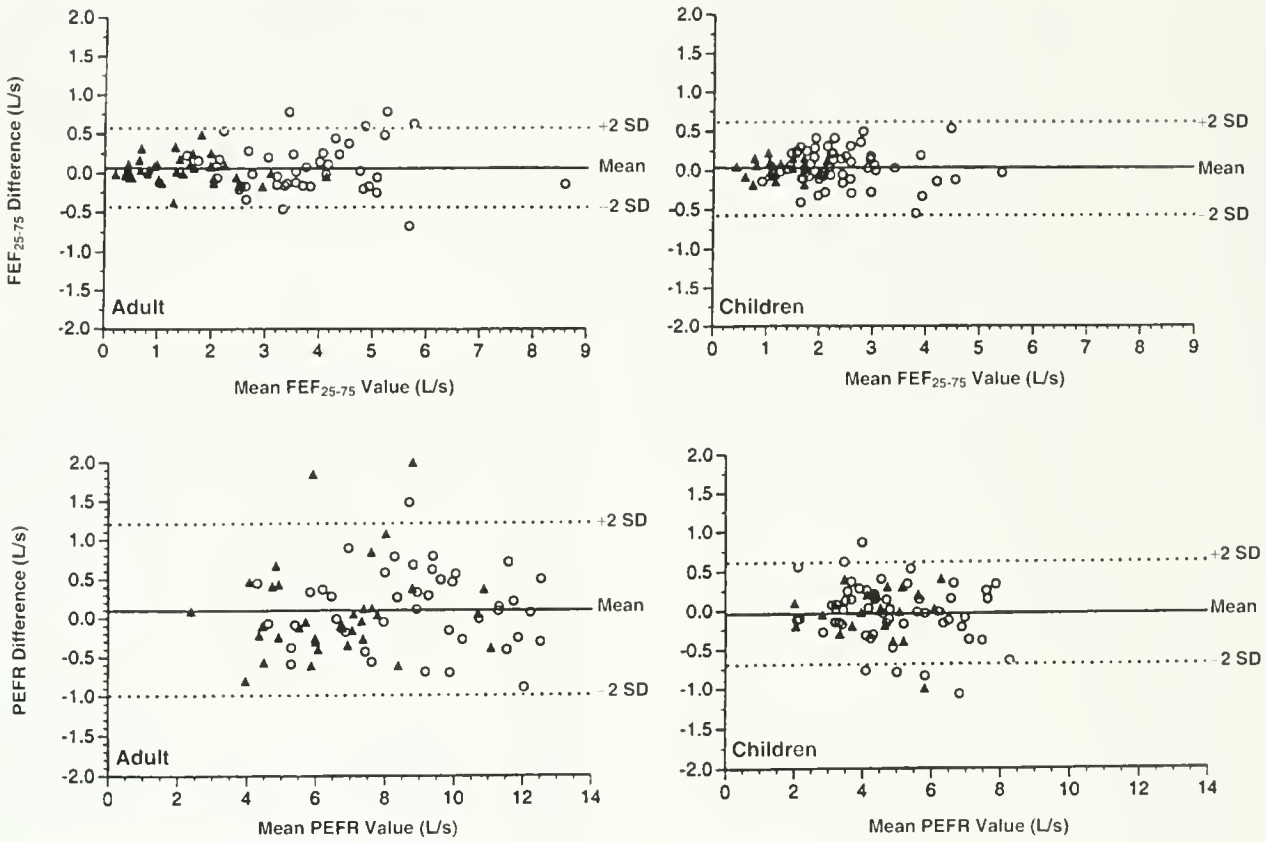

Fig. 2 Each individual difference in PEFR and FEF_{25-75} is plotted against the mean value for that patient. See caption for Fig. 1 for details on the calculations.

Discussion

We found little difference between values for FVC, FEV_1, FEF_{25-75}, or PEFR obtained with and without NC in a large group of children and adults. Several of the differences in the adult values did reach statistical significance in favor of NC, but none of them was of a magnitude that would be considered clinically relevant. Many clinicians and respiratory therapists believe that NC minimize the amount of air that escapes during a forced expiratory effort. However, the data from our study suggest that little air escapes through the nose during a forced maneuver, which is consistent with the findings of Rodenstein and Stanescu.[3]

The number of patients who had clinically important differences was equally split between those who did better with NC and those who had better values w/o NC. Only for FEF_{25-75} was there a trend favoring NC. The definition of a clinically important difference is somewhat arbitrary, and we chose to define it as a >10% change for FVC, FEV_1, and PEFR, and > 20% for FEF_{25-75} based upon the published data concerning the normal variability of these values.[7-12] There is inherent variability in measured spirometric indices with flow measurements being more variable than FVC and FEV_1,[7-9] and this variability is greater for patients with airways obstruction.[10-11] This normal variability makes it difficult to set a specific change that can be considered clinically important. Pennock et al[10] concluded that increases of >10% for FVC, 12% for FEV_1, and 25% for FEF_{25-75} represented significant changes from baseline values.[10] Hegewald et al[12] suggested that in healthy subjects changes in PEFR of 6-8% for adults and 9-10% for children should be considered significant. The relatively large sample size and cross-over design enabled us to detect statistically significant differences in some of the values, but statistical significance should not necessarily be equated with clinical relevance. In our study, the largest difference between values obtained with and w/o NC was for FEF_{25-75} in adult subjects, and it represented only a 2% improvement in favor of NC. The inherent variability of measurements obtained with forced expiratory spirometry is manifest in the distribution of the values in our study. The mean differences were near zero with approximately equal numbers of patients having better values with NC and w/o NC. We propose that the changes

in spirometric values observed in our study were primarily due to the variability of the test rather than to the presence or absence of NC.

We identified only 2 other studies that had evaluated the effect of NC on spirometric measurements. Verral and colleagues[4] found no significant differences between spirometric values obtained with and w/o NC in 136 healthy male volunteers, and Rodenstein and Stanescu[3] also found no significant differences in 20 healthy adult volunteers. The results of our study, which included children and adults with and w/o airways obstruction, agree with these earlier investigations. We found no studies that demonstrated benefit with NC.

It is possible that an occasional patient may lose a clinically important amount of air through the nose when performing spirometry, which could significantly affect measurements. However, the data from our study suggest that the majority of patients do not do this. The most likely explanation for why air does not escape through the nose during forced expiratory spirometry is that the soft palate closes against the posterior pharynx during a forced expiratory maneuver, thus occluding the nasopharynx and blocking nasal air flow. Rodenstein and Stanescu[3] assessed the nasal breathing patterns of 20 normal subjects during various expiratory maneuvers, and all but 1 patient demonstrated complete oral breathing during the maneuvers. Of these subjects, 8 underwent fluoroscopic evaluation while performing the maneuvers, and in all 8 the soft palate was observed to completely occlude the nasopharynx, which resulted in total oral exhalation.

Some patients with anatomic abnormalities of the upper airway may have difficulties in preventing air from escaping through the nose during a forced exhalation. Conditions where this situation may arise include subjects with cleft palate, palatofacial deformity, or who have undergone uvulopalatopharyngoplasty. These patients should always perform spirometry with NC.

Pulmonary function and respiratory therapy technicians should be able to recognize the occasional patient who partially exhales through the nose on forced exhalation. A review of the patient's history and the sound of her or his speech should reveal a palatal abnormality. Listening carefully for nasal exhalation and examining the flow-volume loops for consistency within the FVC trials should help detect further outliers.

Both the ATS[1] and the ERS[2] encourage the use of NC in patients, especially children, during forced expiratory spirometry, but they do not reference studies to support their statements. Our study provides further evidence that NC may not be necessary during forced expiratory spirometry. We suggest that NC should be made optional during forced expiratory spirometry, especially for children. Each patient should perform all forced expiratory maneuvers either with or w/o NC, ie, not alternating the use of NC. We also believe that NC should be used in patients who prefer them, in the occasional patient who demonstrates nasal breathing during the test, and in the patient who has a palatofacial deformity or has undergone uvulopalatopharyngoplasty. Our study evaluated only the effect of NC use during forced expiratory spirometry, so our conclusions pertain solely to that test. Nose clips should still be used for other pulmonary function tests, especially those requiring quiet breathing. The soft palate may not close completely during quiet breathing, potentially allowing more air to escape through the nose. We did not assess the use of NC during forced inspiratory spirometry, though it is unlikely that NC would affect values obtained with this maneuver. We have presented data that should make pulmonary function laboratory personnel reconsider the routine use of NC during forced expiratory spirometry.

ACKNOWLEDGMENTS

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Foreword—Excerpts from the *Expert Panel Report II*

It has been almost exactly 6 years since publication of the first Asthma Guidelines[1] with their 4-pronged approach to effective asthma management:

- Use of objective measures of lung function to assess the severity of asthma and to monitor the course of therapy
- Environmental control measures to avoid or eliminate factors that precipitate asthma symptoms or exacerbations
- Comprehensive pharmacologic therapy for long-term management designed to reverse and prevent the airway inflammation characteristic of asthma as well as pharmacologic therapy to manage asthma exacerbations
- Patient education that fosters a partnership among the patient, his or her family, and clinicians

Because of pharmaceutical breakthroughs and the abundance of literature on the diagnosis and management of asthma, members of the Executive Committee of the National Heart, Lung, and Blood Institute's (NHLBI) National Asthma Education and Prevention Program (NAEPP) decided that it was time for a comprehensive update. More than 5,000 asthma-related abstracts were reviewed by the Expert Panel to determine whether changes and developments since the first Guideline warranted a revision. In addition, the Panel critically reviewed some of the more controversial aspects of asthma diagnosis and management, taking a close look at the quality of study designs and analyzing published studies. This entire process took more than two and a half years and culminated in the publication of an updated report[2] that is a compilation of the latest information about this treatable disease.

The intent of the Guidelines is to serve as a comprehensive guide. Its purpose is to lead or direct a course of action. The Guidelines should not necessarily be considered the absolute and only method to treat and diagnose asthma. The clinician and the patient will need to develop individual treatment plans that are tailored to the specific needs and circumstances of the patient. However, one should be cautioned not to take the Report lightly. A great deal of time, thought, and effort have been invested in this project by some of the world's foremost experts on asthma research and management.

Response from the medical community to the first Report was riddled with both positive and negative reactions. Probably one of the biggest roadblocks to the implementation of the first Guidelines was the failure of primary physicians to 'buy into' them. Several focus group discussions were held to gain a better understanding of why this happened. It was surprising to hear that some feared that they would be held accountable in a court of law if they did not treat exactly as the Guidelines recommended. Some resented what they perceived to be 'cookbook' medicine. These objections must be addressed if primary physicians as 'gatekeepers' of managed care are to embrace the Report II as a tool that helps them make reasonable and sound medical decisions.

Excerpts from Report II that I believe have particular relevance for respiratory care practitioners are published here, but I believe that we need to be conversant with the entire document. What does Report II offer the respiratory care practitioner?—Clear recommendations, a balanced presentation of the evidence for those recommendations, and a rich bibliography supporting them. The complete document can be downloaded from the Internet.

http://www.nhlbi.nih.gov/nhlbi/lung/asthma/prof/asthgdln.htm*

A practical guide that summarizes the 250-page Report will be available this Fall (1997). Copies of the Guideline are available for $20 through NAEPP, (301) 251-1222, NHLBI Information Center, PO Box 30105, Bethesda MD 20824-0105.

Have the Guidelines achieved the goal set for them? Not enough time has elapsed and too few clinicians have been driven to answer this question. Perhaps as more outcomes data are collected, studied, peer reviewed, and published, a definitive answer will evolve. In the meantime, the NAEPP encourages more research aimed at evaluating the impact of implementing the recommendations in this Report.

Thomas J Kallstrom RRT
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National Asthma Education & Prevention Project

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*This site is also available on the AARC home page at http://www.aarc.org

Excerpts from *Expert Panel Report II: Guidelines for the Diagnosis & Management of Asthma*

Pathogenesis and Definition

Key Points

- Asthma, whatever the severity, is a chronic inflammatory disorder of the airways. This has implications for the diagnosis, management, and potential prevention of the disease.
- The immunohistopathologic features of asthma include:
  - Denudation of airway epithelium
  - Collagen deposition beneath basement membrane
  - Edema
  - Mast cell activation
  - Inflammatory cell infiltration
    - Neutrophils (especially in sudden-onset, fatal asthma exacerbations)
    - Eosinophils
    - Lymphocytes (TH2-like cells)
- Airway inflammation contributes to airway hyperresponsiveness, airflow limitation, respiratory symptoms, and disease chronicity.
- Airway inflammation also contributes to several forms of airflow limitation, including acute bronchoconstriction, airway edema, mucus plug formation, and airway wall remodeling. These features lead to bronchial obstruction.
- Atopy, the genetic predisposition for the development of an IgE-mediated response to common aeroallergens, is the strongest identifiable predisposing factor for developing asthma.

Differences From 1991 *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma*

- The critical role of inflammation in asthma has been further substantiated by research. It is recognized that asthma results from complex interactions among inflammatory cells, mediators, and other cells and tissues resident in the airway.
- Evidence indicates that subbasement membrane fibrosis may occur in some patients and that these changes contribute to persistent abnormalities in lung function. The importance of airway remodeling and the development of persistent airflow limitation need further exploration and may have significant implications for the treatment of asthma.

Component 1: Measures of Assessment and Monitoring

Initial Assessment and Diagnosis of Asthma

Key Points

- To establish a diagnosis of asthma, the clinician should determine that:
  - Episodic symptoms of airflow obstruction are present.
  - Airflow obstruction is at least partially reversible.
  - Alternative diagnoses are excluded.
- Recommended mechanisms to establish the diagnosis are:
  - Detailed medical history
  - Physical exam focusing on the upper respiratory tract, chest, and skin
  - Spirometry to demonstrate reversibility
- Additional studies may be considered to:
  - Evaluate alternative diagnoses
  - Identify precipitating factors
  - Assess severity
  - Investigate potential complications
- Recommendations are presented for referral for consultation or care to a specialist in asthma care.

Differences from 1991 *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma*

- Severity classifications were changed from mild, moderate, and severe to mild intermittent, mild persistent, moderate persistent, and severe persistent.
- Examples of questions to use for diagnosis and initial assessment of asthma were added.
- Information on wheezing in infancy and vocal cord dysfunction was expanded in the differential diagnosis section.
- Criteria for referral were refined with input from specialty and primary care physicians.
- More specific recommendations for measuring peak expiratory flow (PEF) diurnal variation are made.

Periodic Assessment and Monitoring: Essential for Asthma Management

Key Points

- The goals of therapy are to:
  - Prevent chronic and troublesome symptoms (eg, coughing or breathlessness in the night, in the early morning, or after exertion)

  - Maintain (near) "normal" pulmonary function
  - Maintain normal activity levels (including exercise and other physical activity)
  - Prevent recurrent exacerbations of asthma and minimize the need for emergency department visits or hospitalizations
  - Provide optimal pharmacotherapy with least amount of adverse effects
  - Meet patients' and families' expectations of and satisfaction with asthma care
- Periodic assessments and ongoing monitoring of asthma are recommended to determine if the goals of therapy are being met. Measurements of the following are recommended:
  - Signs and symptoms of asthma
  - Pulmonary function
  - Quality of life/functional status
  - History of asthma exacerbations
  - Pharmacotherapy
  - Patient-provider communication and patient satisfaction
- Clinician assessment and patient self-assessment are the primary methods for monitoring asthma. Population-based assessment is beginning to be used by managed care organizations.
- Spirometry tests are recommended (1) at the time of initial assessment, (2) after treatment is initiated and symptoms and PEF have stabilized, and (3) at least every 1 to 2 years.
- Patients should be given a written action plan based on signs and symptoms and/or PEF; this is especially important for patients with moderate-to-severe persistent asthma or a history of severe exacerbations.
- Patients should be trained to recognize symptom patterns indicating inadequate asthma control and the need for additional therapy.
- Recommendations on how and when to do peak flow monitoring are presented.

Differences From 1991 *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma*

- The new report includes an additional goal of therapy (meet patients' and families' expectations of and satisfaction with asthma care) that was not listed in the 1991 report
- Periodic assessment of 6 domains of patient health that correspond with the goals of asthma therapy are now recommended, including signs and symptoms, pulmonary function, quality of life, history of exacerbations, pharmacotherapy, and patient provider communication and patient satisfaction.
- The following changes affecting peak flow monitoring have been made:
  - The recommendation for peak flow monitoring was changed from twice daily to morning. If the morning reading is less than 80% of personal best PEF, more frequent peak flow monitoring may be desired.
  - Discussion of inconsistencies in measurement among peak flow meters was added.
  - Use of the individual patient's personal best PEF is emphasized strongly.
- The recommendation for patients at all severity levels to monitor symptoms to recognize early signs of deterioration is emphasized.
- Sample questions to use in periodic assessments were added.

Box 1. Key Indicators for Considering a Diagnosis of Asthma

Consider asthma and performing spirometry if *any* of these indicators are present.* These indicators are not diagnostic by themselves, but the presence of multiple key indicators increases the probability of a diagnosis of asthma. Spirometry is needed to establish a diagnosis of asthma.

- Wheezing—high-pitched whistling sounds when breathing out—especially in children. (Lack of wheezing and a normal chest examination do not exclude asthma.)
- History of any of the following:
  - Cough, worse particularly at night
  - Recurrent wheeze
  - Recurrent difficulty in breathing
  - Recurrent chest tightness
- Reversible airflow limitation and diurnal variation as measured by using a peak flow meter, for example:
  - Peak expiratory flow (PEF) varies 20% or more from PEF measurement on arising in the morning (before taking an inhaled short-acting β-2 agonist) to PEF measurement in the early afternoon (after taking an inhaled short-acting β-2 agonist).
- Symptoms occur or worsen in the presence of:
  - Exercise
  - Viral infection
  - Animals with fur or feathers
  - House-dust mites (in mattresses, pillows, upholstered furniture, carpets)
  - Mold
  - Smoke (tobacco, wood)
  - Pollen
  - Changes in weather
  - Strong emotional expression (laughing or crying hard)
  - Airborne chemicals or dusts
  - Menses
- Symptoms occur or worsen at night, awakening the patient.

*Eczema, hay fever, or a family history of asthma or atopic diseases are often associated with asthma, but they are not key indicators.

Box 2. Importance of Spirometry in Asthma Diagnosis

Objective assessments of pulmonary function are necessary for the diagnosis of asthma because medical history and physical examination are not reliable means of excluding other diagnoses or of characterizing the status of lung impairment. Although physicians generally seem able to identify a lung abnormality as obstructive (Russell et al, 1986), they have a poor ability to assess the degree of airflow obstruction (Shim and Williams, 1980) or to predict whether the obstruction is reversible (Russell et al, 1986).

For diagnostic purposes, spirometry is generally recommended over measurements by a peak flow meter in the clinician's office because there is wide variability even in the best published peak expiratory flow reference values. Reference values need to be specific to each brand of peak flow meter, and such normative brand-specific values currently are not available for most brands. Peak flow meters are designed as monitoring, not as diagnostic, tools in the office (see Component 1—Periodic Assessment and Monitoring). However, peak flow monitoring can establish peak flow variability and thus aid in the determination of asthma severity when patients have asthma symptoms and normal spirometry.

Peak Flow Monitoring

- Patients with moderate-to-severe persistent asthma should learn how to monitor their peak expiratory flow (PEF) and have a peak flow meter at home.
- Peak flow monitoring during exacerbations of asthma is recommended for patients with moderate-to-severe persistent asthma to:
  - Determine severity of the exacerbation
  - Guide therapeutic decisions (see Component 3—Managing Exacerbations and Figure 4-5*) in the home, clinician's office, or emergency department
- Long-term daily peak flow monitoring is helpful in managing patients with moderate-to-severe persistent asthma to:
  - Detect early changes in disease status that require treatment
  - Evaluate responses to changes in therapy
  - Provide assessment of severity for patients with poor perception of airflow obstruction
  - Afford a quantitative measure of impairment
- If long-term daily peak flow monitoring is not used, a short-term (2-3 wk) period of peak flow monitoring is recommended to:
  - Evaluate responses to changes in chronic maintenance therapy

*Editor's Note: The figures, excerpted from the NAEPP Report II, have been rearranged to fit the RESPIRATORY CARE format. However, the original figure numbers have been maintained for reference to the original document. The complete document is available via the internet at:
http://www.nhlbi.nih.gov/nhlbi/lung/asthma/prof/asthgdln.htm
or
http://www.aarc.org

Fig 1-3. Classification of Asthma Severity

Critical Features Before Treatment*	Symptoms†	Nighttime Symptoms	Lung Function
Step 4 Severe Persistent	Continual symptoms Limited physical activity Frequent exacerbations	Frequent	$FEV_1/PEF \leq 60\%$ predicted PEF variability >30%
Step 3 Moderate Persistent	Daily symptoms Daily use of inhaled short-acting β-2 agonist Exacerbations affect activity Exacerbations ≥ 2/wk; may last days	> 1/wk	$FEV_1/PEF > 60\%$ - < 80% predicted PEF variability >30%
Step 2 Mild Persistent	Symptoms > 2/wk but < 1/d Exacerbations may affect activity	> 2/mo	$FEV_1/PEF \geq 80\%$ predicted PEF variability 20-30%
Step 1 Mild Intermittent	Symptoms ≤ 2/wk Asymptomatic and normal PEF between exacerbations Exacerbations brief (from a few hours to a few days); intensity may vary	≤ 2/mo	$FEV_1/PEF \geq 80\%$ predicted PEF variability < 20%

*The presence of one of the features of severity is sufficient to place a patient in that category. An individual should be assigned to the most severe grade in which any feature occurs. The characteristics noted in this figure are general and may overlap because asthma is highly variable. Furthermore, an individual's classification may change over time.

† Patients at any level of severity can have mild, moderate, or severe exacerbations. Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms.

  - Identify temporal relationship between changes in PEF and exposure to environmental or occupational irritants or allergens; it may be necessary to record PEF 4 or more times a day (Chan-Yeung, 1995)
  - Establish the individual patient's personal best PEF
- The Expert Panel does not recommend long-term daily peak flow monitoring for patients with mild intermittent or mild persistent asthma unless the patient/family and/or clinician find it useful in guiding therapeutic decisions. Any patient who develops severe exacerbations may benefit from peak flow monitoring.

Limitations of long-term peak flow monitoring include:
- Difficulty in maintaining adherence to monitoring (Reeder et al, 1990; Chmelik and Doughty, 1994; Malo et al, 1993), often due to inconvenience, lack of required level of motivation, or lack of a specific treatment plan based on PEF
- Potential for incorrect readings related to poor technique, misinterpretation, or device failure

Control of Factors Contributing to Asthma Severity

Key Points
- Exposure of asthma patients to irritants or allergens to which they are sensitive has been shown to increase asthma symptoms and precipitate asthma exacerbations.
- For at least those patients with persistent asthma on daily medications, the clinician should:
  - Identify allergen exposures
  - Use the patient's history to assess sensitivity to seasonal allergens
  - Use skin testing or in vitro testing to assess sensitivity to perennial indoor allergens
  - Assess the significance of positive tests in context of patients' medical history
- Patients with asthma at any level of severity should avoid:
  - Exposure to allergens to which they are sensitive.
  - Exposure to environmental tobacco smoke.
  - Exertion when levels of air pollution are high.
  - Use of β-blockers.
  - Sulfite-containing and other foods to which they are sensitive.
  - Aspirin and nonsteroidal anti-inflammatory drugs if they have a history of sensitivity; if they have severe persistent asthma, they should be counseled regarding the potential risk attendant with use of these drugs.
- Patients should be treated for rhinitis, sinusitis, and gastroesophageal reflux, if present.
- Patients with persistent asthma should be given an annual influenza vaccine.

Differences From 1991 *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma*
- Skin testing or in vitro testing is now specifically recommended for at least those patients with persistent asthma exposed to perennial indoor allergens.
- The use of aspirin and related drugs is not recommended for patients sensitive to aspirin or who have severe persistent asthma, whereas in the 1991 report it was recommended that all asthma patients avoid aspirin.
- Routine use of chemicals to kill house-dust mites and denature the antigen is no longer recommended as a control measure.
- The discussion of tartrazine sensitivity in the 1991 version was deleted.
- Annual influenza vaccinations are now specifically recommended for patients with persistent asthma. The recommendation to consider pneumococcal vaccine was deleted.

Pharmacologic Therapy: The Medications

Overview of the Medications

Key Points: The Medications

Long-term-control medications
- **Corticosteroids:** Most potent and effective anti-inflammatory medication currently available. Inhaled form is used in the long-term control of asthma. Systemic corticosteroids are often used to gain prompt control of the disease when initiating long-term therapy.
- **Cromolyn sodium and nedocromil:** Mild-to-moderate anti-inflammatory medications. May be used as initial choice for long-term-control therapy for children. Can also be used as preventive treatment prior to exercise or unavoidable exposure to known allergens.
- **Long-acting β-2 agonists:** Long-acting bronchodilator used concomitantly with anti-inflammatory medications for long-term control of symptoms, especially nocturnal symptoms. Also prevents exercise-induced bronchospasm (EIB).
- **Methylxanthines:** Sustained-release theophylline is a mild-to-moderate bronchodilator used principally as adjuvant to inhaled corticosteroids for prevention of nocturnal asthma symptoms. May have mild anti-inflammatory effect.
- **Leukotriene modifiers:** Zafirlukast, a leukotriene receptor antagonist, or zileuton, a 5-lipoxygenase inhibitor, may be considered an alternative therapy to low doses of inhaled corticosteroids or cromolyn or nedocromil for patients ≥ 12 years of age with mild persistent asthma, although further clinical experience and study are needed to establish their roles in asthma therapy.

Quick-relief medications
- **Short-acting β-2 agonists:** Therapy of choice for relief of acute symptoms and prevention of EIB.
- **Anticholinergics:** Ipratropium bromide may provide some additive benefit to inhaled β-2 agonists in severe exac-

Fig 3-10. Dosages of Drugs for Asthma Exacerbations in Emergency Medical Care or Hospital

Medications	Dosages		Comments
	Adults	Children	
Inhaled Short-Acting β-2 Agonists			
Albuterol			
Nebulizer solution (5 mg/mL)	2.5-5 mg /20 min for 3 doses, then 2.5-10 mg/1-4 hr as needed, or 10-15 mg/hr continuously needed.	0.15 mg/kg (minimum dose 2.5 mg) every 20 min for 3 doses, then 0.15-0.3 mg/kg up to 10 mg/1-4 hr as needed, or 0.5 mg/kg/hr by continuous nebulization	Only selective β-2 agonists are recommended. For optimal delivery, dilute aerosols to minimum of 4 mL at gas flow of 6-8 L/min.
MDI (90 mcg/puff)	4-8 puffs/20 min up to 4 hr, then every 1-4 hr as needed	4-8 puffs/20 min for 3 doses, then every 1-4 hr as needed	As effective as nebulized therapy if patient is able to coordinate inhalation maneuver. Use spacer/holding chamber.
Bitolterol			
Nebulizer solution (2 mg/mL)	See albuterol dose	See albuterol dose; thought to be half as potent as albuterol on a mg basis	Has not been studied in severe asthma exacerbations. Do not mix with other drugs.
MDI (370 mcg/puff)	See albuterol dose	See albuterol dose	Has not been studied in severe asthma exacerbations.
Pirbuterol			
MDI (200 mcg/puff)	See albuterol dose	See albuterol dose; thought to be one-half as potent as albuterol on a mg basis.	Has not been studied in severe asthma exacerbations.
Systemic (Injected) β-2 agonists			
Epinephrine 1:1000 (1 mg/mL)	0.3-0.5 mg/20 min for 3 doses sq	0.01 mg/kg up to 0.3-0.5 mg/20 min for 3 doses sq	No proven advantage of systemic therapy over aerosol.
Terbutaline (1 mg/mL)	0.25 mg/20 min for 3 doses sq	0.01 mg/kg/20 min for 3 doses then every 2-6 hr as needed sq	No proven advantage of systemic therapy over aerosol.
Anticholinergics			
Ipratropium bromide Nebulizer solution (0.25 mg/mL)	0.5 mg/30 min for 3 doses then every 2-4 hr as needed	0.25 mg/20 min for 3 doses, then every 2 to 4 hr	May mix in same nebulizer with albuterol. Should not be used as first-line therapy; should be added to β-2 agonist therapy.
MDI (18 mcg/puff)	4-8 puffs as needed	4-8 puffs as needed	Dose delivered from MDI is low and has not been studied in asthma exacerbations.
Corticosteroids			
Prednisone Methylprednisolone Prednisolone	120-180 mg/d in 3 or 4 divided doses for 48 hr, then 60-80 mg/d until PEF reaches 70% of predicted or personal best.	1 mg/kg/6 hr for 48 hr then 1-2 mg/kg/d (maximum = 60 mg/d) in 2 divided doses until PEF 70% of predicted or personal best	For outpatient "burst" use 40-60 mg in single or 2 divided doses for adults (children—1-2 mg/kg/d, maximum 60 mg/d) for 3-10 d.

Note: No advantage has been found for higher dose corticosteroids in severe asthma exacerbations, nor is there any advantage for intravenous administration over oral therapy provided gastrointestinal transit time or absorption is not impaired. The usual regimen is to continue the frequent multiple daily dosing until the patient achieves an FEV_1 or PEF of 50% of predicted or personal best and then lower the dose to twice daily. This usually occurs within 48 hr. Therapy following a hospitalization or emergency department visit may last from 3-10 days. If patients are then started on inhaled corticosteroids, studies indicate there is no need to taper the systemic corticosteroid dose. If the followup systemic corticosteroid therapy is to be given once daily, one study indicates that it may be more clinically effective to give the dose in the afternoon at 3 PM with no increase in adrenal suppression (Beam et al, 1992).

erbations. May be an alternative bronchodilator for patients who do not tolerate inhaled β-2 agonists.
- **Systemic corticosteroids:** Used for moderate-to-severe exacerbations to speed recovery and prevent recurrence of exacerbations.

Special Issues Regarding Safety

Short-Acting Inhaled β-2 Agonists

Key Points:
- Short-acting β-2 agonists are the most effective medication for relieving acute bronchospasm.
- Increasing use of short-acting β-2 agonists or the use of more than 1 canister in 1 month indicates inadequate control of asthma and the need for initiating or intensifying anti-inflammatory therapy.
- Regularly scheduled, daily use of short-acting β-2 agonists is generally not recommended.

Long-Acting Inhaled β-2 Agonists

Key Points:
- Long-acting β-2 agonists (salmeterol) can be beneficial to patients when added to inhaled corticosteroid therapy, especially to control nighttime symptoms (Greening et al, 1994; Woolcock et al, 1996). Daily use of long-acting β-2 agonists should generally not exceed 84 mcg (salmeterol; 4 puffs).
- *Salmeterol is not to be used for treatment of acute symptoms or exacerbations.*
- Patient education regarding correct use of salmeterol is critical.
- Patients should be instructed not to stop anti-inflammatory therapy while taking salmeterol even though their symptoms may significantly improve.

Inhaled Corticosteroids

Key Points:
- Inhaled corticosteroids are the most effective long-term therapy available for mild, moderate, or severe persistent asthma; in general, inhaled corticosteroids are well tolerated and safe at the recommended dosages.
- The potential but small risk of adverse events from the use of inhaled corticosteroids is well balanced by their efficacy.
- To reduce the potential for adverse effects, the following measures are recommended:
  - Administer inhaled corticosteroids with spacers/holding chambers.
  - Advise patients to rinse their mouths (rinse and spit) following inhalation.
  - Use the lowest possible dose of inhaled corticosteroid to maintain control.
  - To maintain control of asthma (especially for nocturnal symptoms), consider adding a long-acting inhaled β-2 agonist to a low-to-medium dose of inhaled corticosteroid rather than using a higher dose of inhaled corticosteroid.
  - For children, monitor growth.
  - For postmenopausal women, consider supplements of calcium (1,000 to 1,500 mg/d) and vitamin D (400 units/d). Estrogen replacement therapy, where appropriate, may be considered for patients on doses that exceed 1,000 mcg of inhaled corticosteroid a day.

Pharmacologic Therapy: Managing Asthma Long Term

Key Recommendations for Managing Asthma Long Term
- Persistent asthma is most effectively controlled with daily long-term-control medication, specifically, anti-inflammatory therapy.
- A stepwise approach to pharmacologic therapy is recommended to gain and maintain control of asthma:
  - The amount and frequency of medication is dictated by asthma severity and directed toward suppression of airway inflammation.
  - Therapy should be initiated at a higher level than the patient's step of severity at the onset to establish prompt control and then stepped down.
  - Continual monitoring is essential to ensure that asthma control is achieved.
  - Step-down therapy is essential to identify the minimum medication necessary to maintain control.
- Regular followup visits (at 1- to 6-mo intervals) are essential to ensure that control is maintained and the appropriate step down in therapy is considered.
- Therapeutic strategies should be considered in concert with clinician-patient partnership strategies; education of patients is essential for achieving optimal pharmacologic therapy.
- At each step, patients should be advised to avoid or control allergens, irritants, or other factors that make the patient's asthma worse.
- Referral to an asthma specialist for consultation or co-management of the patient is recommended if there are difficulties achieving or maintaining control of asthma or if the patient requires Step 4 care (see Component 1—Initial Assessment and Diagnosis). Referral may be considered if the patient requires Step 3 care. For infants and young children, referral is recommended if the patient requires Step 3 or 4 care and should be considered if the patient requires Step 2 care.

Fig 3-3. Aerosol Delivery Devices

Device/Drugs	Population	Optimal Technique*	Therapeutic Issues
Metered dose inhaler (MDI) β-2 agonists Corticosteroids Cromolyn sodium and nedocromil Anticholinergics	>5 yr	Actuation during a slow (30 L/min or 3-5 s) deep inhalation, followed by 10-s breathholding. Under laboratory conditions, open-mouth technique (holding MDI 2 in. away from open mouth) enhances delivery to the lung. However it has not consistently been shown to enhance clinical benefit compared to closed-mouth technique (closing lips around MDI mouthpiece).	Slow inhalation may be difficult. Difficulty with coordination of actuation and inhalation, particularly in young children and elderly. Patients may incorrectly stop inhalation at actuation. Deposition of 80% of actuated dose in oropharynx. Mouth washing is effective in reducing systemic absorption (Selroos and Halme, 1991).
Breath-actuated MDI β-2 agonists	> 5 yr	Slow (30 L/min or 3-5 s) inhalation followed by 10-s breathholding.	Indicated for patients unable to coordinate inhalation and actuation. May be particularly useful in elderly (Newman et al, 1991). Slow inhalation may be difficult and patients may incorrectly stop inhalation at actuation. Requires more rapid inspiration to activate than is optimal for deposition. Cannot be used with currently available spacer/holding chamber devices.
Dry powder inhaler (DPI) β-2 agonists Corticosteroids	≥ 5 yr	Rapid (60 L/min or 1-2 s), deep inhalation Minimally effective inspiratory flow is device dependent.	Dose lost if patient exhales through device. Delivery may be MDI depending on device and technique. Can be used in children 4 yr old, but effects are more consistent with children > 5 (Pedersen et al, 1990; Goren et al, 1994; Kemp et al, 1984; Kesten et al, 1994). Most appear to have similar delivery efficiency as MDI either with or without spacer/holding chamber, but some may have delivery > MDI (eg, Turbuhaler) (Thorsson et al, 1994; Agertoft and Pedersen, 1993; Kemp et al, 1989, Melchor et al, 1993; Vidgren et al, 1983). Mouth washing is effective in reducing systemic absorption (Selroos and Halme, 1991).
Spacer/holding chamber	> 4 yr ≤ 4 yr with face mask	Slow (30 L/min or 3-5 s) inhalation or tidal breathing immediately following actuation. Actuation only once into spacer/holding chamber per inhalation (O'Callaghan et al 1994). If face mask is used, allow 3-5 inhalations per actuation (Everard et al, 1992).	Easier to use than MDI alone. With a face mask, enables MDI to be used with small children (Everard et al, 1992; Connett et al, 1993). Simple tubes do not obviate coordinating actuation and inhalation. Bulky. Output may be reduced in some devices after cleaning. The larger volume spacers/holding chambers (> 600 cc) may increase lung delivery over MDI alone in patients with poor MDI technique. The effect of a spacer/holding chamber output from an MDI is dependent on both MDI and spacer type, thus data from one combination should not be extrapolated to all others (Ahrens et al, 1995; Kim et al, 1987). Spacers/holding chambers decrease oropharyngeal deposition and will reduce potential system absorption of inhaled corticosteroid preparations that have higher oral bioavailability (Newman et al, 1984; Brown et al, 1990; Lipworth, 1995; Selroos and Halme, 1991). Spacers/holding chambers are recommended for all patients on medium-to-high doses of inhaled corticosteroids. May be as effective as nebulizer in delivering high doses of β-2 agonists during severe exacerbations.
Nebulizer β-2 agonists Cromolyn Anticholinergics Corticosteroids	≤ 2 yr Patients of any age who cannot use MDI with spacer/holding chamber or spacer and face mask (eg, during exacerbations)	Slow tidal breathing with occasional deep breaths. Tightly fitting face mask for those unable to use mouthpiece	Less dependent on patient coordination or cooperation. Delivery method of choice for cromolyn in children and for high- dose β-2 agonists and anticholinergics in moderate-to-severe exacerbations in all patients. Expensive; time consuming; bulky; output is device dependent and there are significant internebulizer and intranebulizer output variances.

*See figure 4-2 for description of MDI and DPI techniques

Sources: Agertoft and Pedersen 1993; Ahrens et al, 1995; Brown et al, 1990; Connett et al, 1993; Higgins et al, 1987; Crompton and Duncan 1989; Everard et al, 1992; Fuglsang and Pedersen 1986; Goren et al, 1994; Kent et al, 1989; Kesten et al, 1994; Kim et al, 1987; Lipworth 1995, Melchor et al, 1993; Newman et al, 1981, 1984, 1991; O'Callaghan et al, 1994; Pedersen et al, 1990; Pedersen and Mortensen 1990, Prahl and Jenson 1987; Rossing et al, 1980; Ruggins et al, 1993; Schecker et al, 1993; Selroos and Halme 1991; Selroos et al, 1995; Thorsson et al, 1994; Vidgren et al, 1983.

Special Considerations for Managing Asthma in Different Age Groups

Infants and Young Children (5 Years of Age and Younger)

Key Recommendations:

- Diagnosing asthma in infants is often difficult, yet underdiagnosis and undertreatment are key problems in this age group. Thus, a diagnostic trial of inhaled bronchodilators and anti-inflammatory medications may be helpful.
- In general, infants and young children consistently requiring symptomatic treatment more than 2/week should be given daily anti-inflammatory therapy.
- When initiating daily anti-inflammatory therapy, a trial of cromolyn or nedocromil is often given due to the safety profile of these medications.
- Response to therapy should be carefully monitored. Once control of asthma symptoms is established and sustained, a careful step down in therapy should be attempted. If clear benefit is not observed, alternative therapies or diagnoses should be considered.

School-Age Children (Older than 5 Years of Age) and Adolescents

Key Recommendations:

- Pulmonary function testing should use appropriate reference populations. Adolescents compare better to childhood- than to adult-predicted norms.
- When initiating daily anti-inflammatory therapy for mild-to-moderate persistent asthma, a trial of cromolyn or nedocromil is often given.
- Adolescents (and younger children as appropriate) should be directly involved in establishing goals for therapy and developing their asthma management plans.
- Active participation in physical activities, exercise, and sports should be promoted.
- A written asthma management plan should be prepared for the student's school, including plans to ensure reliable, prompt access to medications.

Older Adults

Key Recommendations:

- Chronic bronchitis/emphysema may coexist with asthma. A trial of systemic corticosteroids will determine the presence of reversibility and the extent of therapeutic benefit.
- Asthma medications may aggravate coexisting medical conditions (eg, cardiac disease, osteoporosis); adjustments in the medication plan may need to be made.
- Be aware of increased potential for adverse drug/disease interaction (eg, aspirin, β-blockers).
- Review of patient technique in using medications and devices is essential.

Pharmacologic Therapy: Managing Exacerbations of Asthma

Key Points

- Early treatment of asthma exacerbations is the best strategy for management. Important elements of early treatment include:
  - A written action plan to guide patient self-management of exacerbations at home, especially for patients with moderate-to-severe persistent asthma and any patient with a history of severe exacerbations
  - Recognition of early signs of worsening asthma
  - Appropriate intensification of therapy
  - Prompt communication between patient and clinician about any serious deterioration in symptoms or peak flow, decreased responsiveness to inhaled β-2 agonists, or decreased duration of effect
- Management of asthma exacerbations includes:
  - Inhaled β-2 agonist to provide prompt relief of airflow obstruction
  - Systemic corticosteroids, for moderate-to-severe exacerbations or for patients who fail to respond promptly and completely to an inhaled β-2 agonist, to suppress and reverse airway inflammation
  - Oxygen to relieve hypoxemia for moderate-to-severe exacerbations
  - Monitoring response to therapy with serial measurements of lung function

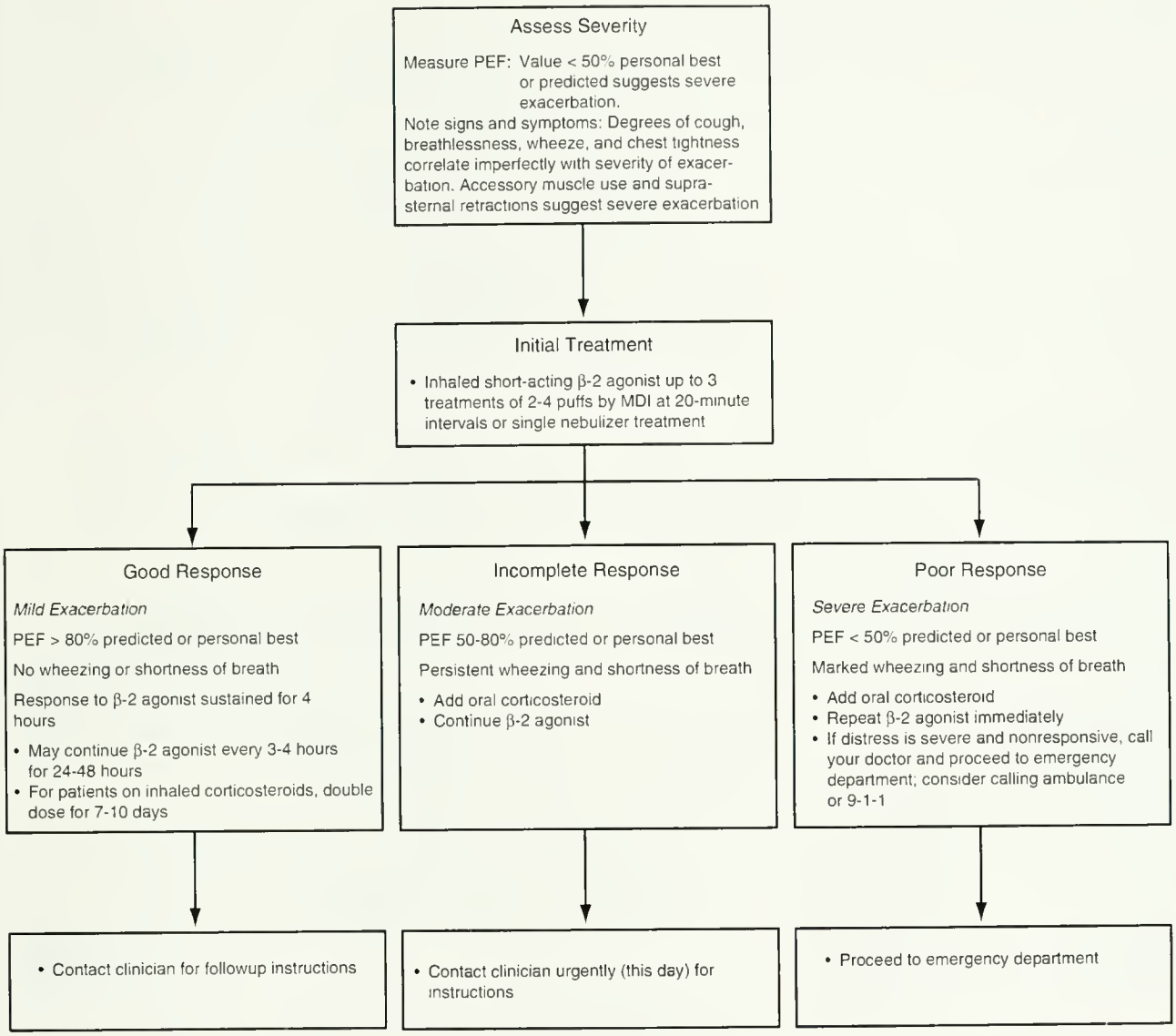

Assess Severity
Measure PEF: Value < 50% personal best or predicted suggests severe exacerbation.
Note signs and symptoms: Degrees of cough, breathlessness, wheeze, and chest tightness correlate imperfectly with severity of exacerbation. Accessory muscle use and suprasternal retractions suggest severe exacerbation
Initial Treatment
• Inhaled short-acting β-2 agonist up to 3 treatments of 2-4 puffs by MDI at 20-minute intervals or single nebulizer treatment
Good Response
Mild Exacerbation
PEF > 80% predicted or personal best
No wheezing or shortness of breath
Response to β-2 agonist sustained for 4 hours
• May continue β-2 agonist every 3-4 hours for 24-48 hours
• For patients on inhaled corticosteroids, double dose for 7-10 days
Incomplete Response
Moderate Exacerbation
PEF 50-80% predicted or personal best
Persistent wheezing and shortness of breath
• Add oral corticosteroid
• Continue β-2 agonist
Poor Response
Severe Exacerbation
PEF < 50% predicted or personal best
Marked wheezing and shortness of breath
• Add oral corticosteroid
• Repeat β-2 agonist immediately
• If distress is severe and nonresponsive, call your doctor and proceed to emergency department; consider calling ambulance or 9-1-1
• Contact clinician for followup instructions
• Contact clinician urgently (this day) for instructions
• Proceed to emergency department

Fig. 3-8. Management of asthma exacerbations through home treatments. Patients at high risk of asthma-related death should receive immediate clinical attention after initial treatment. Additional therapy may be required.

Fig 3-9. Classifying Severity of Asthma Exacerbations*

	Mild	Moderate	Severe	Respiratory Arrest Imminent
Symptoms				
Breathlessness	While walking	While talking (infant—softer, shorter cry; difficulty feeding)	While at rest (infant—stops feeding)	
	Can lie down	Prefers sitting	Sits upright	
Talks in	Sentences	Phrases	Words	
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Signs				
Respiratory rate†	Increased	Increased	Often > 30/min	
Use of accessory muscles; suprasternal retractions	Usually not	Commonly	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate, often only end expiratory	Loud; throughout exhalation	Usually loud; throughout inhalation and exhalation	Absence of wheeze
Pulse/min‡	< 100	100-120	>120	Bradycardia
Pulsus paradoxus	Absent < 10 mm Hg	May be present 10-25 mm Hg	Often present >25 mm Hg (adult) 20-40 mm Hg (child)	Absence suggests respiratory muscle fatigue
Functional Assessment				
PEF % predicted or % personal best	> 80%	Approximately 50-80%	< 50% predicted or personal best or response lasts < 2 hr	
P_{aO_2} (on air)	Normal (test not usually necessary)	> 60 mm Hg (test not usually necessary)	< 60 mm Hg: possible cyanosis	
and/or P_{aCO_2}	< 42 mm Hg (test not usually necessary)	< 42 mm Hg (test not usually necessary)	≥ 42 mm Hg: possible respiratory failure (see text)	
S_{aO_2}% (on air) at sea level	> 95% (test not usually necessary)	91-95%	< 91%	
	Hypercapnia (hypoventilation) develops more readily in young children than in adults and adolescents.			

*Note: The presence of several parameters, but not necessarily all, indicates the general classification of the exacerbation. Many of these parameters have not been systematically studied, so they serve only as general guides.
† Guide to normal rates of breathing in awake children: < 2 mo— < 60/min, 2-12 mo— < 50/min, 1-5 yr— < 40/min, 6-8 yr— < 30/min.
‡ Guide to normal pulse rates in children: 2-12 mo— < 160/min, 1-2 yr— < 120/min, 2-8 yr— < 110/min.

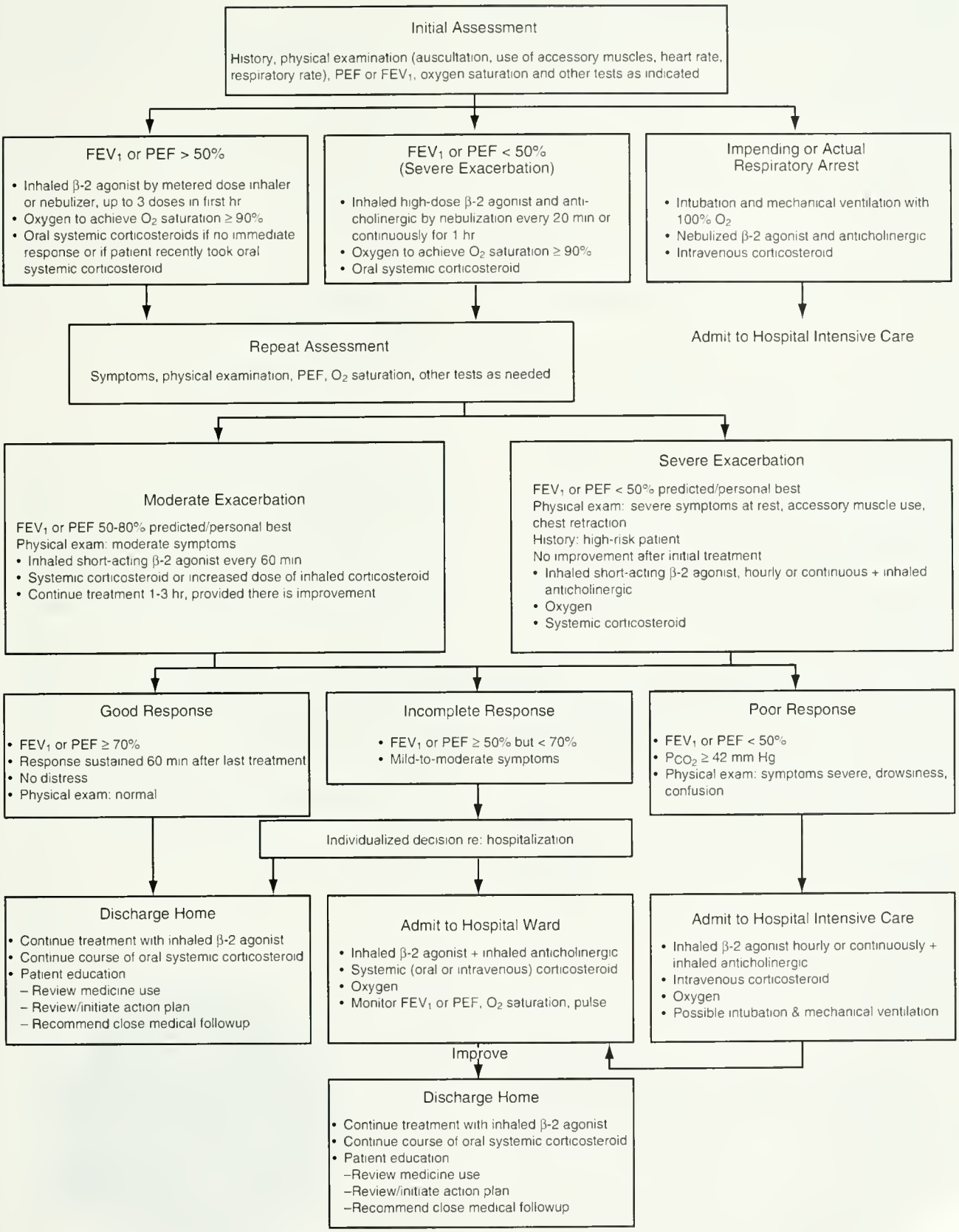

Initial Assessment
History, physical examination (auscultation, use of accessory muscles, heart rate, respiratory rate), PEF or FEV1, oxygen saturation and other tests as indicated
FEV1 or PEF > 50%
• Inhaled β-2 agonist by metered dose inhaler or nebulizer, up to 3 doses in first hr
• Oxygen to achieve O2 saturation ≥ 90%
• Oral systemic corticosteroids if no immediate response or if patient recently took oral systemic corticosteroid
FEV1 or PEF < 50% (Severe Exacerbation)
• Inhaled high-dose β-2 agonist and anticholinergic by nebulization every 20 min or continuously for 1 hr
• Oxygen to achieve O2 saturation ≥ 90%
• Oral systemic corticosteroid
Impending or Actual Respiratory Arrest
• Intubation and mechanical ventilation with 100% O2
• Nebulized β-2 agonist and anticholinergic
• Intravenous corticosteroid
Admit to Hospital Intensive Care
Repeat Assessment
Symptoms, physical examination, PEF, O2 saturation, other tests as needed
Moderate Exacerbation
FEV1 or PEF 50-80% predicted/personal best
Physical exam: moderate symptoms
• Inhaled short-acting β-2 agonist every 60 min
• Systemic corticosteroid or increased dose of inhaled corticosteroid
• Continue treatment 1-3 hr, provided there is improvement
Severe Exacerbation
FEV1 or PEF < 50% predicted/personal best
Physical exam: severe symptoms at rest, accessory muscle use, chest retraction
History: high-risk patient
No improvement after initial treatment
• Inhaled short-acting β-2 agonist, hourly or continuous + inhaled anticholinergic
• Oxygen
• Systemic corticosteroid
Good Response
• FEV1 or PEF ≥ 70%
• Response sustained 60 min after last treatment
• No distress
• Physical exam: normal
Incomplete Response
• FEV1 or PEF ≥ 50% but < 70%
• Mild-to-moderate symptoms
Poor Response
• FEV1 or PEF < 50%
• PCO2 ≥ 42 mm Hg
• Physical exam: symptoms severe, drowsiness, confusion
Individualized decision re: hospitalization
Discharge Home
• Continue treatment with inhaled β-2 agonist
• Continue course of oral systemic corticosteroid
• Patient education
– Review medicine use
– Review/initiate action plan
– Recommend close medical followup
Admit to Hospital Ward
• Inhaled β-2 agonist + inhaled anticholinergic
• Systemic (oral or intravenous) corticosteroid
• Oxygen
• Monitor FEV1 or PEF, O2 saturation, pulse
Admit to Hospital Intensive Care
• Inhaled β-2 agonist hourly or continuously + inhaled anticholinergic
• Intravenous corticosteroid
• Oxygen
• Possible intubation & mechanical ventilation
Improve
Discharge Home
• Continue treatment with inhaled β-2 agonist
• Continue course of oral systemic corticosteroid
• Patient education
–Review medicine use
–Review/initiate action plan
–Recommend close medical followup

Fig. 3-11. Management of asthma exacerbations: emergency department and hospital-based care.

Fig 3-12. Hospital Discharge Checklist for Patients with Asthma Exacerbations

Intervention	Dose/Timing	Education/Advice	MD/RN Initials
Inhaled medications (MDI + spacer/holding chamber	Select agent, dose, and frequency (eg, albuterol)	Teach purpose Teach technique	
β-2 agonist	2-6 puffs q 3-4 hr prn	Emphasize need for spacer/holding chamber	
Corticosteroids	Medium dose	Check patient technique	
Oral medications	Select agent, dose, and frequency (eg, prednisone 20 mg bid for 3-10 d)	Teach purpose Teach side effects	
Peak flow meter	Measure AM and PM PEF and record best of 3 tries each time	Teach purpose Teach technique Distribute peak-flow diary	
Followup visit	Make appointment for followup care with primary clinician or asthma specialist	Advise patient (or caregiver) of date, time, and location of appointment within 7 d of hospital discharge	
Action plan	Before or at discharge	Instruct patient (or caregiver) on simple plan for actions to be taken when symptoms, signs, and PEF values suggest recurrent airflow obstruction	

The complete NAEPP Report II document is available via the internet at:

http://www.nhlbi.nih.gov/nhlbi/lung/asthma/prof/asthgdln.htm

or

http://www.aarc.org

See the Notices Section in this issue for listings of other Helpful Web Sites of interest to medical professionals

Patient Satisfaction Is an Indicator of Quality of Care

Introduction

There are those who would make a case for the premise that the only thing that matters to society in health care today is decreased mortality associated with an acceptable quality of life at a price we can afford. Dedicated clinicians may quickly label that premise as overly simplistic and wonder and worry about assuring (and monitoring and measuring) quality of care in the face of cost-saving and restructuring frenzy. More and more we, as health care providers, are made aware of the human factors that affect the quality of the care that we deliver and one of those factors is the importance of patient and family perceptions—patient satisfaction, if you will.

We do not know exactly what our health care system will look like in the future; but, whatever the financial structure and care delivery model, one aspect is already clear: quality of care will no longer be taken for granted. All providers will be required to monitor and improve it.

Quality of Care

Demands of Proof of Quality

The demand for proof of quality is already growing. The Health Care Financing Administration (HCFA) has been making mortality figures public for several years. Some states are now making additional hospital outcome and care-related statistics available to the public. Business coalitions with regional health care purchasing clout are forcing providers to reveal similar data. In addition, the Joint Commission for Accreditation of Healthcare Organizations (JCAHO) has been disseminating previously sacrosanct information on individual hospital standards of performance.[1]

Many health care reform advocates place great importance on providing comparative health care quality information to the public, both to help create better informed consumers and purchasers and to make providers more attentive to quality improvement and customer satisfaction. Health care report cards, including customer satisfaction results, will provide comparative information intended to help the consumer make various types of health care related choices, particularly choice of health care plan or hospital.[2] Report card advocates say that consumers and businesses purchasing health care are demanding information on quality and performance measures.[3]

JCAHO requires health care organizations to monitor patient satisfaction as an indicator of the quality of care and to guide quality improvement. The specific JCAHO standard[4] states that the assessment of how well processes are designed and how well they operate must include information from patients, families, staff members, and others. This includes their needs and expectations, perceptions of how the hospital could improve and perceptions of how well the hospital performs relative to the dimensions of performance. The hospital may collect this information in several ways. Satisfaction surveys, staff suggestion boxes, and regularly scheduled meetings with patients, families, payors, and staff provide opportunities to gain input on performance and opportunities for improvement.[4]

Leaders in other industries—hotels, airlines, express delivery couriers, and other service businesses—have long recognized that quality and satisfaction were not only commendable but also essential for success. In health care, this notion has been embraced and given greater emphasis only in recent years.

The Relationship between Satisfaction & Quality

Several factors have prompted renewed interest in the relationship between patient satisfaction and health care quality: a re-emergence of interest in the basic subject of quality, an increase in the attention being paid to outcome and "clinical indicators," implementation of the concepts of Total Quality Management (TQM) and Continuous Quality Im-

provement (CQI), and greater recognition that patient satisfaction is an important measure of health "system" performance and outcome. There is a growing consensus among patients and their families, health professionals, local, state and federal regulators, and national accreditation organizations, that satisfaction with care may be one of our most important measures of quality health care.[5] Patient satisfaction can be divided into several major categories: (1) satisfaction with the care experience, (ie, process of delivery), (2) satisfaction with the health benefit derived from the care experience (ie, outcome), (3) overall sentiments of satisfaction with the total care experience, and (4) personal cost-benefit analysis of the goodness of process and outcomes. Factors in any of these categories may contribute to future behaviors (eg, return use, recommendations).[1,5] Satisfaction not only leads patients to continue using the health care services but also decreases the incidence of malpractice suits.[6] Survey instruments must provide specific information on what customers need in order to chart process improvement plans and meet the goals of TQM and CQI.

According to interviews with patients who had undergone bypass surgery at the University of Wisconsin Hospitals, Madison, the most common comment was that patients were surprised to wake up on a ventilator. "The feeling of panic that patients expressed about that experience was tremendous." They said that "no one had ever told them to expect that."[7] Another study suggested that the quality of medical care for patients with asthma or COPD can be improved by adjusting provided care to the needs expressed by patients.[8] Patients who are ill are extremely concerned about how quickly staff respond to their needs. Consumers make judgments about quality based on variables they can appraise: courtesy, responsiveness, attentiveness, and perceived competence of staff.[9] Clinical medicine is built upon an interaction between persons seeking assistance (ie, patients) and persons offering assistance—physicians and other members of the health care team. Educational programs have increasingly focused on developing and assessing medical students and residents' patient-interaction skills.[10]

The Importance of Perception

Service quality perceptions in hospitals can be improved through improving communication between patients and providers. The literature suggests that a primary positioning strategy used to attain a sustainable competitive advantage for service organizations, such as those in the health care industry, is through the provision of best-in-the-world service quality and customer satisfaction in the minds of consumers.[11] Whether the business is a restaurant or health care organization, one cannot do business without knowing the feelings of the customer.

Patients judge the quality of care from their perspective. To achieve an image of quality, health care executives must commit to and effectively manage customer perceptions as well as clinical quality. Although one might argue that patients are incapable of judging the quality of their care and, perhaps, don't know 'what is good for them,' patients are either satisfied or they are not. Patient satisfaction is more than an indicator, it is also a component of care. When patients are satisfied, a host of additional factors impact on care, including: enhancement of trust leading to greater compliance, increased tolerance of uncomfortable or frightening interactions, lessening of stress, and increased tolerance of delays. Satisfied patients will have enhanced healing and treatment effectiveness, complain less, be more likely to return, and be less likely to initiate lawsuits.[1,12] Attention to patient satisfaction is good business and, simply, the 'right thing' to do.

This is the new age of health care consumerism. Researchers continue to collect outcome data and launch sweeping surveys in the less tangible area of consumer satisfaction.[13] Marketing-based consumer research has traditionally emphasized customer satisfaction as a dominant concern in the period immediately following the purchase of goods or services. The emerging health services literature suggests this orientation is particularly prevalent in today's health care environment.[14]

Patients and potential patients (ie, family and friends) harbor perceptions about an institution, its various services, and its workers. These perceptions drive patients' decisions to select, use, and return to the institution.[15] Satisfaction surveys used in health care organizations typically focus on the reactions of patients to the care they have received, ignoring the role of family and friends as influences and potential consumers of the service themselves. We believe that this is a mistake because dissatisfaction among a patient's family and friends can present serious consequences for a health care organization through the loss of future business. In a study conducted over a 6-year period, hospital inpatients gave higher satisfaction ratings for their medical care and services than did their family members and friends. The returns for decreasing negative word-of-mouth effects can range from $6,000 to $400,000.[16]

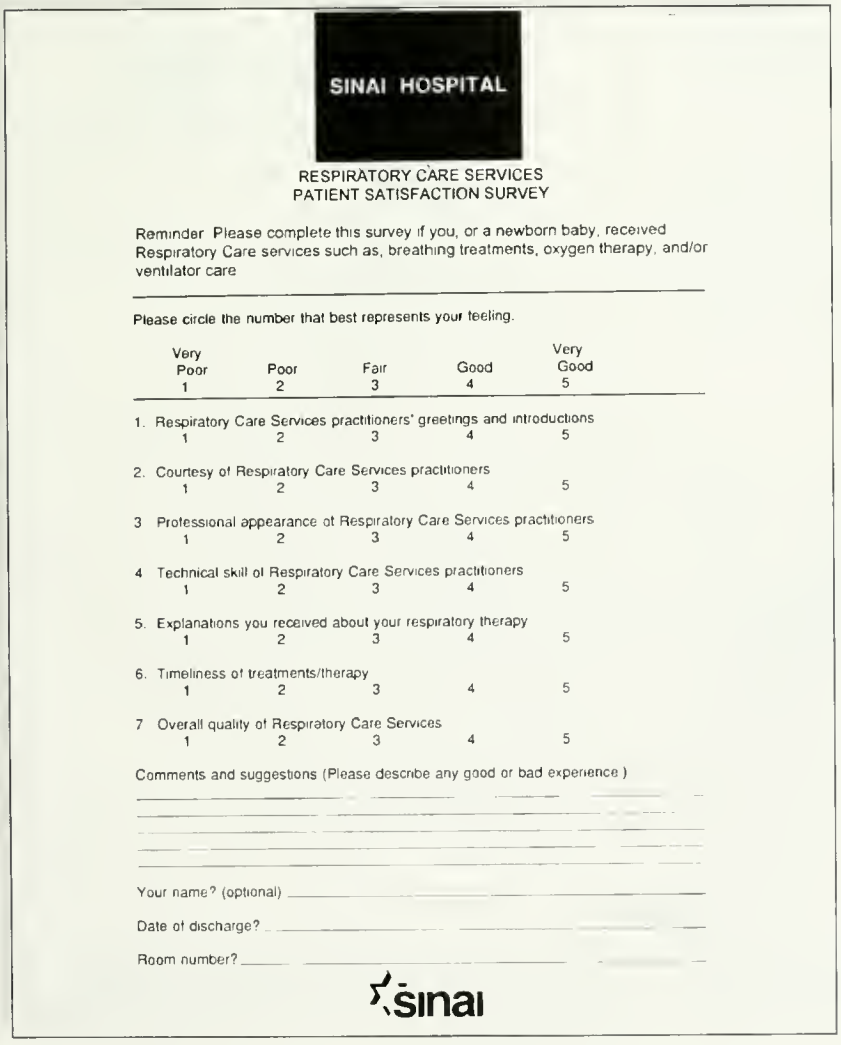
Today, the issue of quality is on the agenda of every hospital and health care provider in the country. Customer perceptions about the quality of an organization can mean the difference between success and failure of the organization. Health care executives today cannot afford to ignore customer perceptions. From the customer's perspective, satisfaction with the hospital experience is highly correlated with quality of care.[17] The single most important action hospital executives can take to maintain quality from the patient's perspective is to deliver a satisfactory experience. In other words, meet, and better yet, exceed the customer's expectations. The concern shown for patients well-being will be the single most important issue on which hospitals' overall quality of care will be judged.[16]

What Sinai Has Done

Sinai Hospital is a 598-bed, not-for-profit, acute care hospital, which is now a part of the Detroit Medical Center, an integrated academic health facility affiliated with Wayne State Medical School, the third largest medical school in the U.S. Respiratory care services are provided hospitalwide by 56 members, including 39 registered therapists, 9 CRTT, registry-eligible therapists, 1 CRTT, and 3 credential-eligible practitioners. Therapists interact with patients, families, and health care personnel in the emergency department, the critical care units including a neonatal unit, the pulmonary/ventilator-dependent patient unit, general care units, the pulmonary function laboratory, and the out-patient cystic fibrosis clinic.

Although we realize that assuring patient satisfaction is only one aspect of TQM and CQI, Sinai Respiratory Care Services has been surveying patient satisfaction with respiratory care services for more than 10 years. The instrument in current use (Fig. 1) incorporates 7 points, a Likert-type rating scale, and space for narrative comments. Patients are asked to rate their level of satisfaction regarding: RCP greeting and introduction, courtesy, professional appearance, technical skill, and explanations about therapy and timeliness. In addition, patients are asked to rate the overall quality of respiratory care services. During the early years of use, the surveys were distributed to inpatients by staff therapists (sometimes reluctantly) and collected by them prior to patient discharge. The rate of return of surveys was low, and we believed that responses were not as candid and forthright as they might have been had the surveys been returned anonymously by mail.

Although "response selectivity" may be an issue in mailed surveys (ie, individuals with little writing ability and those who have no interest in the subject may not respond) and response rates are usually higher with telephone surveys,[18] we have come to believe that mailed questionnaires generate a wider range of responses and minimize the "acquiescence bias" that personal and telephone interviews tend to elicit. Self-administered questionnaires are generally cheaper, but



SINAI HOSPITAL

RESPIRATORY CARE SERVICES
PATIENT SATISFACTION SURVEY

Reminder Please complete this survey if you, or a newborn baby, received Respiratory Care services such as, breathing treatments, oxygen therapy, and/or ventilator care

Please circle the number that best represents your feeling.

	Very Poor	Poor	Fair	Good	Very Good
	1	2	3	4	5
1. Respiratory Care Services practitioners' greetings and introductions	1	2	3	4	5
2. Courtesy of Respiratory Care Services practitioners	1	2	3	4	5
3 Professional appearance of Respiratory Care Services practitioners	1	2	3	4	5
4 Technical skill of Respiratory Care Services practitioners	1	2	3	4	5
5. Explanations you received about your respiratory therapy	1	2	3	4	5
6. Timeliness of treatments/therapy	1	2	3	4	5
7 Overall quality of Respiratory Care Services	1	2	3	4	5

Comments and suggestions (Please describe any good or bad experience.)

Your name? (optional) _____

Date of discharge? _____

Room number? _____

sinai

Fig. 1. Patient satisfaction survey distributed for Respiratory Care Services at Sinai Hospital, Detroit, Michigan.

also may be more appropriate for dealing with especially sensitive issues, if the survey offers anonymity.[18]

When the hospital employed a national firm (Press, Ganey Associates Inc, South Bend IN) to survey and analyze customer satisfaction hospitalwide, Respiratory Care Services was, of course, included.

Press-Ganey's service to its clients includes the survey instrument and quarterly reports in which the results of site data examination across time are compared to a national database.[19] This comparison allows clients to target aspects of the care process that need improvement by comparison and benchmarking with a national peer group.[20] The so-called face and predictive validity of Press-Ganey data are supported by published research.[2,3,18] We believe that using an outside vendor enhances data quality and credibility.

However, the Press-Ganey survey includes only one question specific to respiratory care (Fig. 2), and we wanted more feedback than answers to the single question afforded. Fortunately for our department, we are able to have our specific instrument included with the hospitalwide services instrument when it is mailed. We now have the advantages of a mailed, anonymous survey with recordkeeping and data analysis by a reputable firm. Results of our patient satisfaction surveys are compiled, presented to the department each month (Fig. 3) and quarterly, and integrated into the department quality/performance improvement plan.

For a 20-month sampling period (January 1993-August 1994) during which respiratory care staff members distributed and collected surveys, 381 surveys were returned for an average of 19/month. For the first 16 months of mailed surveys (September 1994-December 1995), 852 surveys were returned for an average of 53/month—an increase in surveys/month of 179%.

In Conclusion

Our business is health care and health care in this country is in institutional transition.[21] Quality is sometimes defined as doing things right, on time, the first time, and meeting patients' needs and expectations. Compliments from patients are good and may signal areas of strong performance. Complaints point out areas and aspects of care that need improvement. To improve quality, managers and practitioners must view performance deficits as opportunities for improvement. Using the assessment of satisfaction and patient experience as an indicator of quality of care is important. The current literature is rich with excellent ideas about how satisfaction and quality of care can be joined. We have found that our survey results provide valuable feedback to the department as a monitor of customer satisfaction and identifies opportunities for improvement. It has been our experience that mailing patient satisfaction surveys significantly increases survey return over RCP distribution. We believe that benchmark goals for patient satisfaction ratings should be established and incor-

Please circle the number that best represents your feeling.

	Very Poor	Poor	Fair	Good	Very Good
	1	2	3	4	5
10 Nuclear Medicine	1	2	3	4	5
11 Pharmacists/Pharmacy	1	2	3	4	5
12 Recovery Room staff (if you had surgery)	1	2	3	4	5
13. Respiratory Care Services	1	2	3	4	5

(If you received Respiratory Care during your stay, please complete the enclosed sheet and fold it inside the main survey when you return it.)

Comments (describe good or bad experience).

_____

_____

_____

_____

1. VISITORS AND FAMILY

1	Courtesy of the people at the information desk	1	2	3	4	5
2	Valet Parking	1	2	3	4	5
3	Security	1	2	3	4	5
4	Adequacy of visiting hours	1	2	3	4	5
5	Courtesy of telephone operators	1	2	3	4	5
6	Adequacy of signs in hospital	1	2	3	4	5
7	Accommodations and comfort for visitors	1	2	3	4	5
8	Nursing attitudes toward your visitors	1	2	3	4	5
9	Information given your family about your condition and treatment	1	2	3	4	5
10	Visitor's rating of the hospital cafeteria/coffee shop	1	2	3	4	5

Comments (describe good or bad experience)

_____

_____

_____

Fig. 2. Excerpt from hospitalwide patient satisfaction survey (Coyright Press, Ganey Associates Inc, reprinted with permission).

Sinai Hospital

Survey: Respiratory Care Services/Pt. Satisfaction Survey
Covers Surveys: From 2/01/96 to 2/29/96
No. of Questionnaires: 27

Division: All
Location: All

Questions		Responses						
		Very Poor	Poor	Fair	Good	Very Good		
1. Greetings and Introductions	Percent			3.7	18.5	77.8	Weighted Score	4.74
	Number			1	5	21	Total Responses	27
							Standard Deviation	2.5338
2. Courtesy of Practitioners	Percent				26.9	73.1	Weighted Score	4.73
	Number				7	19	Total Responses	26
							Standard Deviation	2.5004
3. Professional Appearance	Percent				25.9	74.1	Weighted Score	4.74
	Number				7	20	Total Responses	27
							Standard Deviation	2.5192
4. Technical Skill	Percent			3.8	15.4	80.8	Weighted Score	4.77
	Number			1	4	21	Total Responses	26
							Standard Deviation	2.5888
5. Explanations about Therapy	Percent			4.2	16.7	79.2	Weighted Score	4.75
	Number			1	4	19	Total Responses	24
							Standard Deviation	2.5536
6. Timeliness of Treatments	Percent			4.2	20.8	75.0	Weighted Score	4.71
	Number			1	5	18	Total Responses	24
							Standard Deviation	2.4776
7. Overall Quality	Percent				23.1	76.9	Weighted Score	4.77
	Number				6	20	Total Responses	26
							Standard Deviation	2.5739
							Average Score for Section	4.74

Room #	Patient Comments
7409	Respiratory was very good and satisfactory.
	Everything was fine. My stay was good, for me and to me.
3110	Had two practitioners come by my room. First person came too soon after surgery—my husband knew the procedure. Couple days later, a second practitioner did come and reassure me of procedure and was aware first visit may need follow up. Very helpful.
1122	They were very informative about what was happening and what they were doing at the time.
3822	The hospital is very nice and caring.
7426	Very professional.

Sinai

Fig. 3. Sample of monthly patient satisfaction report compiled by Sinai Hospital Patient Representative Department.

porated into departmental performance improvement and appraisal programs.

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Post-Myocardial Infarction Drugs: Respiratory Implications

Hugh S Mathewson MD and Judith A Mathewson MS RRT

Acute myocardial infarction (MI) is the chief cause of death in the United States and in most developed countries.[1] Although primary prevention and treatment[2] have steadily improved, the annual incidence in this country still approaches 500,000 cases. Almost half of all victims die before they reach the hospital, about 7-15% die during hospitalization, and another 7-15% die during the following year.[3] With the aging population, MI has become a public health problem of increasing magnitude.

For the survivors of MI, a primary concern is prevention of a subsequent episode. Numerous studies have shown that adjunctive drug therapy, particularly with β-adrenergic blocking agents and angiotensin converting enzyme (ACE) inhibitors, is effective in preventing recurrent MI.[4] Another important consideration is prevention of sudden death from cardiac dysrhythmia. If there is persistent ventricular irritability following infarction, a continuous antiarrhythmic drug regimen is indicated.

Rapidly growing numbers of patients, especially middle-aged men who have an MI history, are taking maintenance doses of cardiovascular drugs. Many of these agents can affect the bronchopulmonary system; it is these untoward actions that are of interest and importance in respiratory care. The effects of cardiovascular drugs on the lungs and airways have been recently reviewed by Zitnik.[5,6] A list of these drugs and their therapeutic indications are shown in the accompanying table.

β-adrenergic receptor blockers are strongly advocated for all patients with MI who do not have definite contraindications. Intravenous administration, followed by oral therapy, should be instituted in hospitalized patients within 24 hours after the onset of symptoms. If tolerated, the drugs should be continued for at least 2 or 3 years, and perhaps longer.

Clear contraindications to the use of β-blockers in post-MI patients are drug-induced bronchospasm and pulmonary edema. Although β-1 blockade is the therapeutic goal, considerable β-2 antagonism occurs with some drugs of this class, especially with the prototype agent *propranolol*. Nonselective β-blockers cause dose-dependent decreases in expiratory flows, and worsen bronchospasm in asthmatic patients.[7] Increased airway obstruction may accompany chronic obstructive pulmonary disease (COPD). In rare instances, β-blockers have caused the development of pulmonary fibrosis or a lupus-like syndrome with lung infiltrates.

Efforts have been made to develop compounds that are β-1 selective; the best examples to date are *metoprolol*[8] and *atenolol*.[9] These agents, the partial agonist *pindolol*,[10] and the mixed α-β antagonist *labetalol*[11] are better tolerated by patients with asthma or COPD. Of special interest is *esmolol*, an ultrashort-acting β-blocker suitable for emergency intravenous use. It is usually well tolerated by asthmatics; if not, its bronchospastic effect will probably be dissipated in a few minutes.[12]

Table. Cardiovascular Drugs Given during and after Myocardial Infarction[4-6]

β-adrenergic blocking agents—anti-ischemic, antihypertensive, antiarrhythmic.
- Propranolol (generic, Inderal)
- Metoprolol (Lopressor)
- Atenolol (Tenormin)
- Pindolol (generic, Visken)
- Timolol (generic, Blocadren)
- Labetolol (Normodyne, Trandate)
- Esmolol (Brevibloc)

Angiotensin-converting enzyme (ACE) inhibitors—reduce left ventricular dysfunction, slow progression to congestive heart failure.
- Captopril (Capoten)
- Enalapril (Vasotec)
- Enaprilat (Vasotec Injection)
- Lisinopril (Prinivil, Zestril)
- Ramipril (Altace)
- Fosinopril (Monopril)

Antiarrhythmics—prevent life-threatening dysrhythmias.
- Sodium channel blockers
  - Lidocaine (generic, Xylocaine)
  - Tocainide (Tonocard)
  - Mexiletine (Mexitil)
  - Encainide (no longer available in USA)
  - Flecainide (Tambocor)
  - Procainamide (generic, Pronestyl, others)
  - Moricizine (Ethmozine)
- Sodium and potassium channel blockers
  - Amiodarone (Cordarone)
  - Sotalol (Betapace)

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ACE inhibitors are now universally recommended during MI, and for a period of 5 to 6 weeks following onset.[13,14] If the patient has residual left ventricular dysfunction, such as an ejection fraction of less than 40% or symptoms of congestive heart failure, the therapy should be continued for at least 3 years.[4] ACE inhibitors are notorious for causing a chronic nonproductive cough. The incidence is variously reported as 5-15%, and appears to have about the same frequency of occurrence with all such compounds commonly used.[15] The onset of cough often follows 1 to 2 months after initiation of treatment. There has been concern that ACE inhibitors increase the risk of bronchospasm,[16] but this is rarely observed, and the present consensus is that the drugs are safe to use in asthmatic patients.[17]

The etiology of ACE inhibitor-induced cough is obscure, but has been attributed to blockade of the metabolism of bronchial irritant neuropeptides, such as substance P and bradykinin.[6] As one might assume, there is greater susceptibility to cough in patients with increased upper airway irritability. Withdrawal of the drug usually results in resolution of cough in a few weeks. When ACE inhibitor therapy must be continued, the cough may subside spontaneously or may respond to treatment with cromolyn.[18]

A more serious condition attributable to ACE inhibitors is angioneurotic edema, which may terminate fatally. However, only a few such cases have been reported.[6] A newly approved drug, *losartan*, which is a direct acting angiotensin-II receptor antagonist, does not appear to induce cough or cause angioneurotic edema.[19]

Antiarrhythmic drugs are essential to prevent sudden death during and following acute MI. Between 4 and 18% of patients have potential ventricular fibrillation within the first 48 hours. Among all patients with sudden cardiac death, 21-27% are found to have had acute MI at the time of death, and 44 to 52% have had prior infarction.[20] In the first year post-MI, sudden death accounts for 30 to 60% of all mortality.[21] Ventricular tachycardia or fibrillation accounts for 75% of deaths,[22] and mortality is increased fourfold in patients with more than 10 premature ventricular contractions per hour.[23]

The collective experience with oral antiarrhythmic drugs has not been altogether successful. Because *lidocaine* can be given only parenterally, its effects are largely confined to acute treatment in the hospital. Longer acting congeners of lidocaine given post-MI have proven ineffective or provocative of toxic cardiac or systemic side effects. Trial series using *encainide* and *flecainide* were terminated because of failure of significant benefit or increased mortality.[24] *Tocainide*[25] and *mexiletine*[26] have been associated with a pneumonitis/fibrosis syndrome.

Similar experiences have been reported with *moricizine*[27] and *sotalol*,[28] two antiarrhythmic compounds unrelated to lidocaine.

Amiodarone is a potent antiarrhythmic agent that is of especial interest in prevention of sudden death.[29] It is an iodinated compound that reportedly reduces post-MI mortality.[30] However, it has multiple pathogenic effects involving several organ systems. About 5% of patients on maintenance medication with this drug develop the pneumonitis/fibrosis syndrome, either in subacute or chronic form.[31,32] The subacute form may mimic infectious pneumonia, with fever, chest pain, and the appearance of lung infiltrates. The chronic form is characterized by cough, dyspnea, and weight loss. A restrictive ventilatory effect is evident on pulmonary function testing. Amiodarone has also been implicated in episodes of acute noncardiogenic pulmonary edema with development of respiratory distress syndrome.[33] Some of these occurrences have followed uneventful surgical procedures; it is suggested that high oxygen tensions administered during anesthesia may be contributory.[34]

The pneumonitis/fibrosis syndrome can have a fatal termination. Immediate discontinuance of the offending antiarrhythmic agent and corticosteroid therapy are essential to successful resolution.[6]

Drug-induced lupus is characterized by fever and arthralgias and may progress to a destructive generalized vasculitis. *Procainamide*, often prescribed for suppression of ventricular dysrhythmias, is the drug most often associated with this disorder.[35] Pulmonary infiltrates and pleural effusion are the manifestations of respiratory system involvement. Procainamide is biotransformed by acetylation, a mechanism whose rate is genetically determined. Subjects who are congenitally slow acetylators are more susceptible to lupus than subjects who are fast acetylators.[36] Discontinuance of the drug usually results in permanent remission, but corticosteroid therapy may be required on occasion.

Aspirin should be mentioned as an established deterrent to heart attacks. It is a cyclo-oxygenase inhibitor in the biosynthesis of arachidonic acid derivatives, prostaglandins, and notably thromboxanes which prevent platelet aggregation and coronary thrombus formation.[37] Although chronic respiratory complications from aspirin are uncommon, there is a definite incidence of bronchospasm often attributed to allergy. An alternative hypothesis is that aspirin-induced bronchospasm is caused by diversion of arachidonic acid metabolism into the lipoxygenase pathway, with increased formation of leukotrienes.[38]

To summarize, it is now advocated that β-adrenergic antagonists and ACE inhibitors be administered during acute MI, and for extended periods thereafter, along with aspirin, to reduce the risk of reinfarction. Although implantable defibrillators are placed in patients at high risk of sudden death, the long-term use of antiarrhythmic drugs is widespread. All these compounds have the potential to cause untoward bronchopulmonary effects. The respiratory care practitioner will encounter post-MI patients with increasing frequency, and probably will be confronted more often with the respiratory symptoms and signs elicited by cardiovascular drugs.

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Using Waveforms To Optimize Inspiratory Rise Time during Pressure Support Ventilation

M Dee Johnson MS CPFT RRT, James J Lawson BS RRT, and Robert S Campbell RRT

Case Summary

A 74-year-old white woman involved in a motor vehicle accident was transported by air to the emergency department. On admission, the patient was conscious, followed commands, and complained of severe chest and abdominal pain. Vital signs were heart rate 128/min, blood pressure 90/palpable, and respiratory rate 36/min. Analysis of arterial blood drawn with the patient receiving oxygen via a non-rebreathing mask at 15 L/min revealed pH 7.28, P_{aCO_2} 35 torr, P_{aO_2} 138 torr, and S_{aO_2} 0.98. Auscultation revealed diminished breath sounds bilaterally. An anteroposterior chest radiograph made with portable equipment showed a 15% pneumothorax and multiple rib fractures on the right with associated flail chest. A chest tube was placed on the right side with an escape of air noted on insertion. Peritoneal lavage was grossly positive for blood, and the patient was immediately transported to the operating room for an exploratory laparotomy.

Laparotomy revealed multiple liver lacerations, which were repaired and packed in the operating room. Fractures of her left tibia and fibula were stabilized. Blood loss was estimated at 4,500 mL, and 17 units of packed red blood cells and many units of platelets and fresh frozen plasma were administered.

Following surgery, the patient was transferred to the surgical intensive care unit (SICU) with stable vital signs (heart rate 118/min; blood pressure of 105/58). Anesthesia was not intentionally reversed, and she was placed on a Siemens 300 Ventilator (Siemens Life Support Systems, Iselin NJ) with V_T 800 mL, synchronized intermittent mandatory ventilation (SIMV) rate of 10 breaths/min, positive end-expiratory pressure (PEEP) 5 cm H_2O, and F_{IO_2} 0.60. Blood gas analysis revealed pH 7.32, P_{aCO_2} 38 torr, P_{aO_2} 102 torr, and S_{aO_2} 0.98.

The patient's postoperative course was complicated by failure to wean from mechanical ventilation. Over the course of the next 7 days, the patient was slowly weaned to 15 cm H_2O of pressure support (PS) but failed her daily unassisted breathing trials with F_{IO_2} of 0.35, due to tachypnea.

Part 1

On Postoperative Day 9, a freestanding respiratory monitor (Bicore CP 100 Pulmonary Monitor, Allied Healthcare Products Inc, Riverside CA) was added to the patient-ventilator system to provide additional information during weaning. The patient remained on the 300 ventilator in the spontaneous mode with a PEEP/CPAP 5 cm H_2O, PS 15 cm H_2O, and F_{IO_2} of 0.35. The ventilator was set to provide flow triggering with an inspiratory rise time (IRT) setting of 10% with the CMV breath rate set to 6 breaths/min. Figure 1 illustrates the waveforms at these settings.

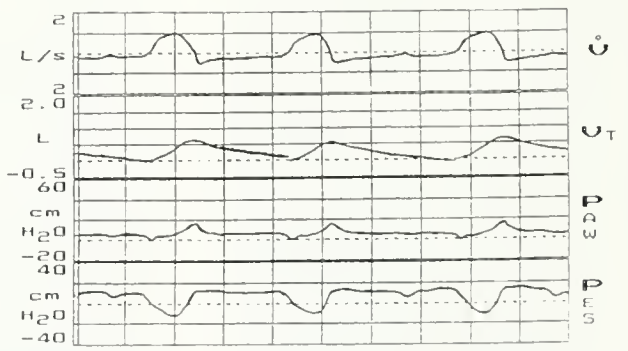

Fig. 1. Waveform as recorded by the freestanding respiratory monitor on Postoperative Day 9. Inspiratory rise time setting is 10%

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How would you answer these questions?

How would you describe the inspiratory flow waveform?

What is suggested by the expiratory flow waveform? What are some of the possible contributing factors that should be evaluated?

What is suggested by the airway pressure waveform?

What is suggested by the esophageal pressure waveform? What are some possible corrective measures?

Part 2

Following analysis of the waveforms in Figure 1, the IRT was decreased to 5% in an effort to improve ventilator flow response and patient-ventilator synchrony. Figure 2 illustrates the waveforms at these settings.

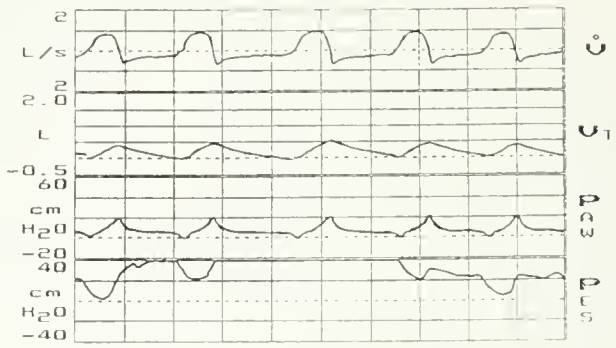

Fig. 2. Waveform resulting from decrease in inspiratory rise time to 5%.

How would you answer these questions?

What effect (if any) did the change in IRT have on the inspiratory flow waveform?

What effect (if any) did the change in IRT have on the expiratory flow waveform?

What effect (if any) did the change in IRT have on the airway pressure waveform?

What effect (if any) did the change in IRT have on the esophageal pressure waveform?

What are possible causes of the elevated esophageal pressure in the center of this waveform?

Part 3

The IRT was further reduced to 1% with all other settings remaining constant. Figure 3 illustrates the waveforms at these settings.

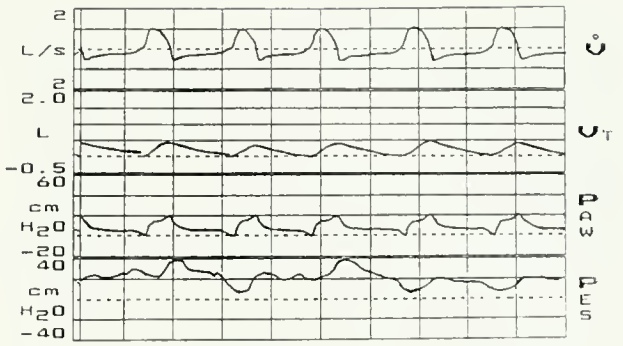

Fig. 3. Waveform resulting from decrease in inspiratory rise time to 1%.

How would you answer these questions?

What effect (if any) did the change in IRT have on the inspiratory flow waveform?

What effect (if any) did the change in IRT have on the expiratory flow waveform?

What effect (if any) did the change in IRT have on the airway pressure waveform?

What effect (if any) did the change in IRT have on the esophageal pressure waveform?

Answers and Discussion on Page 524

Answers, Part 1

Inspiratory Flow. The inspiratory flow waveform is sinusoidal in shape with a peak inspiratory flow of 51 L/min.

Expiratory Flow. Two potential problems are suggested by the expiratory flow waveform. First, expiratory flow does not return to zero prior to the onset of inspiration. This suggests that the patient is 'air-trapping' resulting in intrinsic PEEP. Measured intrinsic PEEP at this time was 5 cm H_2O. Intrinsic PEEP may be the result of increased airways resistance[1] (from bronchospasm or secretions), imposed resistance[2] (endotracheal tube, humidifier, and breathing circuit), increased minute ventilation requirements,[3] or inappropriate ventilator settings. Measured airways resistance (R_{aw}) was 24 cm $H_2O \cdot s \cdot L^{-1}$ at this time. The patient's bronchodilator therapy frequency was changed from every 4 hours to every 2 hours in an effort to decrease airways resistance, and indirect calorimetry ruled out overfeeding and excessive $\dot{V}_{CO_2}$ as the cause of the high minute ventilation (11.9 L). Second, a notch in the expiratory flow waveform prior to the first breath and between the second and third breaths may indicate patient inspiratory effort that is not sensed by the ventilator. This may be due to the intrinsic PEEP, which has been previously reported to have a negative effect on ventilator triggering.[4]

Airway Pressure. During inspiration, airway pressure does not exceed baseline pressure until the middle of the breath and the set pressure support level is not achieved. Measured peak inspiratory pressure is 16 cm H_2O. The airway pressure spike at the end of inspiration may be due to the low terminal flow criterion (5% of peak flow), which is inherent to the ventilator. This delay in pressure rise suggests that patient flow demand exceeds the flow output of the ventilator in the early phase of inspiration.

Esophageal Pressure. There is a large negative deflection during the inspiratory phase with a measured peak negative esophageal pressure of −12 cm H_2O and a total change in pressure of 24 cm H_2O. This suggests that the patient work of breathing is increased. Measured patient work of breathing (WOBpt) at this time is 1.8 J/L. Patient work of breathing may be reduced by increasing the pressure support level,[5-8] adding mandatory breaths, or manipulating the IRT.[9,10]

Answers, Part 2

Inspiratory Flow. The inspiratory flow waveform continues to be sinusoidal in shape. The peak inspiratory flow is now 56 L/min. The peak inspiratory flow is achieved earlier in the inspiratory phase as compared to Figure 1.

Expiratory Flow. Expiratory flow still does not return to zero prior to the onset of inspiration, suggesting the continued presence of intrinsic PEEP. Measured intrinsic PEEP is 5 cm H_2O. There are no patient efforts that are unrecognized by the ventilator.

Airway Pressure. The rise in airway pressure occurs earlier and is steeper than seen in Figure 1. The set PS level is achieved at the end of inspiration. There is no pressure plateau during the inspiratory phase. The steeper pressure rise indicates that flow output of the ventilator is higher than that seen in Figure 1, but remains inadequate as evidenced by the lack of pressure plateau during inspiration. An airway pressure spike is visible as in Figure 1.

Esophageal Flow. The negative deflection in esophageal pressure is less than that seen in Figure 1. The absolute change in esophageal pressure is 16 cm H_2O. Measured WOBpt at this time was 1.5 J/L. The elevated esophageal pressure seen in this waveform does not appear to be associated with the patient's breathing pattern. If the increased esophageal pressure were to occur during the expiratory phase, it could be attributed to active exhalation. More commonly, and as is seen here, the increased esophageal pressure occurs independent of the breathing pattern and is most likely the result of swallowing. One other explanation may be a Valsalva maneuver by the patient.

Answers, Part 3

Inspiratory Flow. The initial inspiratory flow waveform now 'ramps up' faster, with a measured peak inspiratory flow of 69 L/min.

Expiratory Flow. The expiratory flow appears to reach zero (or near zero) prior to inspiration on the first 3 breaths in Figure 3. Measured intrinsic PEEP is 2 cm H_2O at this time.

Airway Pressure. The rise in airway pressure is much steeper, the PS level is achieved earlier in the inspiratory phase, and there is a pressure plateau present. The airway pressure spike visible in Figures 1 and 2 is still present.

Esophageal Pressure. The peak negative esophageal pressure deflections are improved and the absolute change in esophageal pressure is lower. The change in esophageal pressure is 9 cm H_2O. Measured WOBpt is 0.55 J/L at this setting.

Discussion

Altering flow during PS was first described by Branson et al[9] in 1990. Proper adjustment of the flow during PS resulted in improved patient-ventilator synchrony and reduced WOBpt. Since that time, several ventilator manufacturers have incorporated a control for adjusting the initial flow of PS breaths. This case report demonstrates the benefit of

manipulating the initial flow and the importance of monitoring airway graphics in determining the optimal initial flow setting during PS.

Clinician adjustment of initial flow during PS is currently available on the (1) Bear 1000 (Bear Medical Corporation, Division of Allied Medical. Riverside CA);(2) Evita I and IV (Dräger Critical Care Systems, Chantilly VA); (3) Quantum (Healthdyne Technologies, Marietta GA); and (4) Siemens 300. The Newport Wave (Newport Medical Instruments, Newport Beach CA) ventilator uses an algorithm to determine the initial flow of PS breaths, and adjusts the initial flow 'on the fly' in response to changing patient demand (see Reference 12 for detailed description). The initial flow may be indirectly set on the Siemens 900C ventilator by adjusting the working pressure.[11]

The initial flow output determines the rate of rise in airway pressure. Higher flows result in a steep (vertical) pressure rise, while lower flows result in a more prolonged ('ramped') rise in pressure. Most manufacturers allow clinicians to adjust an independent and arbitrary control that ranges from a fastest setting to a slowest setting.

Siemens labels this control "Inspiratory Rise Time %" (IRT) on the 300 ventilator. The IRT setting on the 300 determines the period during which pressure increases to the set PS level following patient initiation of a breath. The range is from 0% to 10% and the percentage is based on the ventilatory cycle time (t_{cycle}). The t_{cycle} is derived from the formula: $t_{cycle} = 60/\text{CMV rate}$. [For example, if the CMV rate is set at 10 breaths/min, the $t_{cycle} = 6$ s. With the IRT set to 10%, it would take 0.6 s to achieve the PS level; if the IRT were changed to 1%, it takes 0.06 s to achieve the PS level resulting in a higher (more aggressive) initial flow output. If the CMV rate is then changed to 6 breaths/min ($t_{cycle} = 10$ s), an IRT of 1% results in a 0.01-second period to achieve the PS level, again increasing the initial flow output.] Clinicians should be aware that the IRT (time to reach PS) is partly determined by the CMV rate control because of its effect on t_{cycle}. The IRT setting determines the time to reach the set inspiratory pressure. The effect of manipulating the IRT is a change in the initial flow output of the ventilator. An IRT of 1% results in a higher initial flow output than an IRT of 10%.

In this patient, an IRT of 10% resulted in increased WOBpt because the inspiratory flow output of the ventilator did not match (was lower than) patient demand. This was not obvious to the clinician by clinical assessment or by monitoring provided on the 300 ventilator. It was not until the pulmonary monitor was placed that patient-ventilator dyssynchrony was recognized. Use of graphics allowed us to optimize the flow during PS, lower the patient's WOBpt, and improve her synchrony with the ventilator without increasing the level of ventilatory support.

The optimal setting of IRT may be determined by analysis of the airway pressure waveform. An IRT setting that results in an initial flow that is too high may be evidenced by 'pressure overshoot' in the early phase of inspiration (Fig. 4). The IRT may also effect the inspiratory time and volume delivery of PS breaths. Initial flows in excess of patient demand create a steeper flow deceleration and may cause premature termination of inspiration. Conversely, when the initial flow output of the ventilator is lower than patient demand, the rise in airway pressure may be delayed or prolonged, or in some cases may prohibit attaining the set PS level (Fig. 1). In our opinion, the optimal IRT setting results in an airway pressure waveform that has a minimal negative pressure deflection at the onset of inspiration, a rapid rise to set PS level while avoiding pressure overshoot, and presence of a short pressure plateau prior to termination of inspiration (Fig. 3).

As evidenced by this case report, the IRT is an integral and important setting that should be carefully adjusted to the individual needs of each patient. The inspiratory flow demand of each patient is determined by inherent resistance and compliance characteristics and respiratory drive. There is no one setting that could be considered 'best' for all patients or 'best' for the same patient over time.

Use of graphics allowed us to better diagnose, make appropriate adjustments, and monitor patient response to ventilator adjustments. Improvements were noted initially on the pressure-time waveforms and corroborated with the measured improvements in WOBpt and intrinsic PEEP. The additional information made available to us by use of the freestanding monitor allowed us to better determine the optimal dose of inhaled bronchodilator while measuring patient response (airways resistance). Additionally, we were able to titrate the level

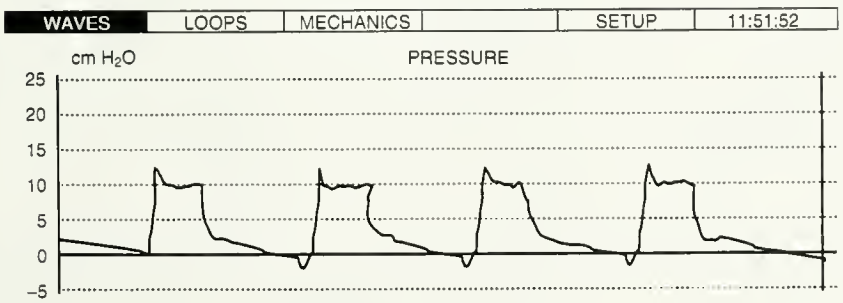

Fig. 4. 'Pressure overshoot' resulting from an initial flow that is too high.

of ventilatory support in an attempt to strengthen the respiratory muscles while avoiding fatigue. The patient had a tracheostomy tube placed on Day 15 and was liberated from mechanical ventilation on Day 18.

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CORRECTION: ATTRIBUTION OF FIGURES

Attribution of Figures 1, 2, and 3 in Wilson BG. Extracorporeal and Intracorporeal techniques for the treatment of severe respiratory failure. Respir Care 1996;41(4):306-317 was inadvertently omitted.

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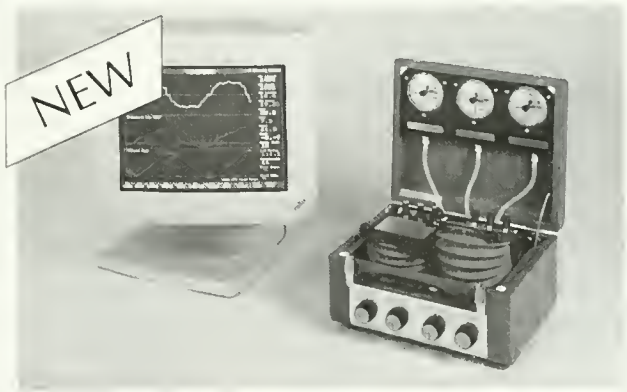

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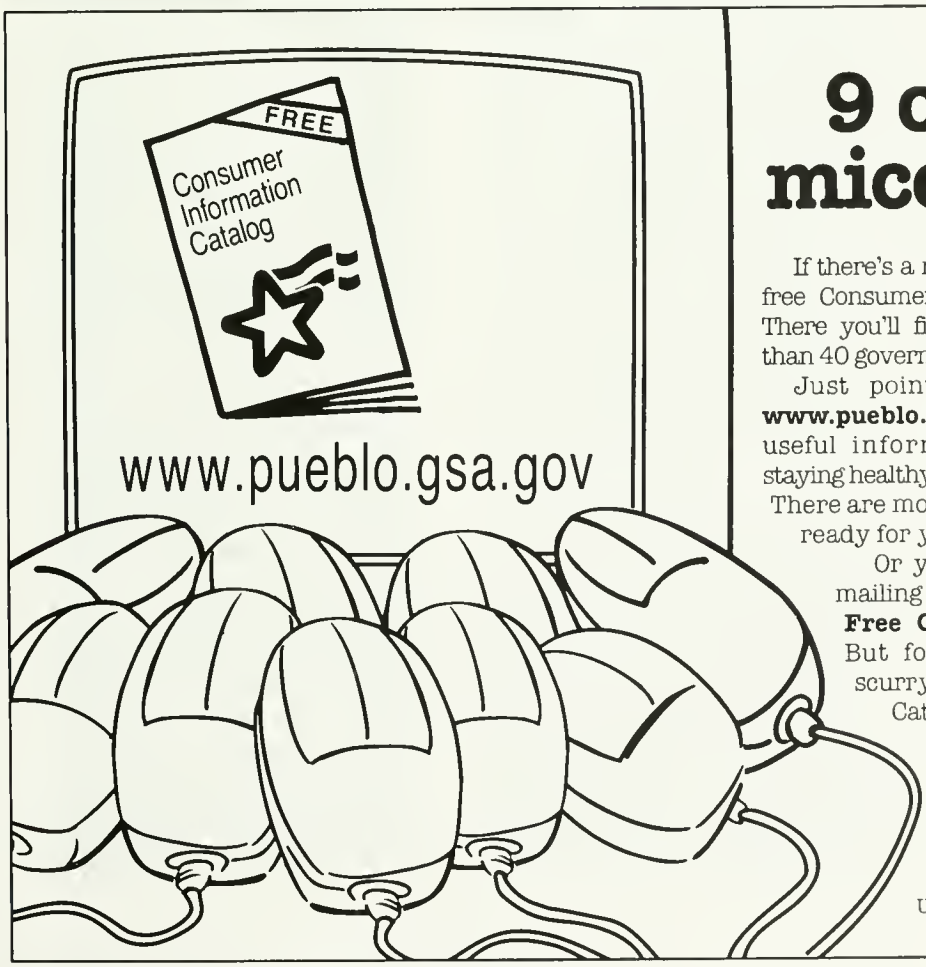

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Respiratory Care Drug Reference by AJ McLaughlin Jr and SR Levine. Softcover, 383 pages. Gaithersburg MD: Aspen Publishers Inc, 1997. $46.

Mosby's Respiratory Care Drug Reference by JL Rau Jr. Softcover, 352 pages. St Louis: Mosby, 1997. $25.95.

The pharmacology literature is replete with handbooks for rapid reference that detail selected drugs. The 2 books reviewed here are intended for use by the respiratory care practitioner (RCP). Both are primarily drug dictionaries with the compounds listed in alphabetical order, in contrast to pharmacology texts in which drugs are categorized according to dominant therapeutic action. The authors of both books have attempted to circumscribe the classes of drugs selected for inclusion based on their use in cardiopulmonary medicine.

The McLaughlin and Levine work is larger in size and content, approaching the scope of a nursing drug manual. It is designed to be used both in the hospital and in home care. An introductory section (Part I) is a series of short reviews of some drug classes of special importance to the RCP. However, adrenergic drugs are conspicuously absent. The main body of the compendium (Part II) is a generic list of virtually all compounds of importance in cardiorespiratory medicine. Each drug entry includes the trade names, therapeutic class, pregnancy risk category, indications, dosage and routes of administration, and the preparations available. Accompanying each description is a special note, "Respiratory Care Considerations," which describes the adverse reactions and contraindications of particular importance to the RCP. About 600 different drugs are reviewed.

Four appendices are added. The first lists the schedules of controlled substances in the U.S.A. and Canada. The second defines the pregnancy risk categories as specified by the Food and Drug Administration. The third is an alphabetical list of pulmonary and cardiovascular disease conditions with the drugs commonly employed for their treatment. The fourth appendix is a short but up-to-date bibliography. The index shows the generic drug names in boldface, in contrast to the trade names.

The book by Rau is smaller (it fits comfortably in the side pocket of a lab jacket) and more specialized in content. Also, it is more directed to hospital respiratory care. Inside the front cover the classes, generic and trade names, and dosages of aerosolized drugs used by the RCP are tabulated. The body of the work is divided into 5 sections.

Section I is a useful description of aerosol delivery devices. Section II is a brief summary of the uses of aerosolized agents. Section III is a review of the aerosol drugs themselves, describing the indications and uses, interactions, and adverse effects of the principal agents identified in Section II. Section IV describes several groups of drugs that are important in the management of the critically ill and highlights the considerations especially appropriate in respiratory care. These groups include anti-infective agents, antidepressants, antipsychotics, sedative-hypnotics, local and general anesthetic agents, and opioids. Some newer drugs in these classes are not included (midazolam, desflurane, sevoflurane, and fluoxetine and other serotonin reuptake inhibitors), and there is no mention of neuromuscular blocking agents. Midazolam is subsequently described; however, some of the drugs listed do not appear in the index.

Section V comprises more than 80% of the book, and is a compendium of drugs used for patients with cardiopulmonary disease or in critical care. About 250 drugs are described, including indications and uses, routes of administration, pharmacokinetic data, adverse effects, interactions, contraindications, and precautions. These descriptions parallel those in nursing guides, but a section on respiratory care considerations accompanies each entry. A few recently introduced drugs are absent (salmeterol, zafirlukast).

The book concludes with 6 appendices: units of measure, systems of measurement, drug dose calculation for adults and for children, a table of drug-induced pulmonary disorders, and a nomogram for estimation of body surface area.

Both of these works can be recommended to RCPs who are active in patient care. Although lacking in the descriptive detail of pharmacology texts, they provide portable, accessible sources of basic drug information that offer worthwhile convenience to the busy practitioner. Students will appreciate the brevity and conciseness of the drug descriptions, coupled with the comments and suggestions appropriate to respiratory management.

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Prehospital Emergency Care—A Guide for Paramedics, Third edition, by J Abbott and MJ Gifford. Softcover, 369 pages. New York, NY: The Parthenon Publishing Group, 1996. $23.95.

This book, written by two emergency physicians, is divided into nine logically arranged chapters. They include Prehospital Patient Assessment, Medical, Pediatric, Trauma, and Environmental Treatment Protocols, Hazardous Materials Protocols, Prehospital Procedures—Basic and Advanced, Prehospital Medications, and Operational Procedures.

Up front, I need to say that I am biased against the use of pulse oximetry in the field. Many pulse oximeters have not been validated for use in areas of high vibration and movement. Their use adds another piece of equipment that most likely will not yield any more data for field diagnosis or change the treatment protocols for a patient, and it may divert the caregiver's attention from truly important things (and perhaps may result in treatment of the monitor and not the patient). We do not carry pulse oximeters on our ambulance, and I have never said "I wish I had a pulse oximeter."

With this background, I have to take issue with the authors' emphasis on the use of pulse oximetry and their repeated statement that one should "titrate to pulse oxime-

try of > 90% if possible." Because hypoxia is the worst thing that can happen to a patient (as Haldane observed, it "not only stops the machine, it wrecks the machinery"), if a practitioner suspects hypoxia of any organ or the body as a whole, the highest concentration of oxygen available should be administered. In the presence of chest pain or head trauma, the S_{aO_2} may be normal even without the administration of supplemental oxygen. I suspect one could find few if any who would find fault with oxygen administration at these times. What, then, is the point of measuring pulse oximetry in these circumstances? Besides, it is probably not possible to administer too much oxygen in the prehospital setting. However, the authors do avoid the common trap of hesitating to administer sufficient oxygen to the patient with known chronic obstructive pulmonary disease. They advocate increasing the oxygen as needed and assisting ventilation if necessary.

There are scattered throughout the text some excellent clinical tips and words of wisdom. Under prehospital assessment, for instance, the authors advocate using the radio as an assessment tool to share the picture of the patient with the physician. Documentation of dysrhythmias is stressed, not only for help in treatment of the patient but also for use as a learning tool for other prehospital care providers. The section covering traumatic shock is full of clinical 'pearls.' In addition, the authors stress the point that hypotension is a late sign of hypovolemic shock and that elderly patients can actually be hypotensive with a systolic pressure greater than 90 mm Hg. And, they make the not-so-obvious point that someone with a relative bradycardia may be taking beta blockers.

A fact new to me was that spinal cord injury above the level of T-8 removes tenderness, guarding, and rigidity as clues to abdominal injury. Of use to those living on the seashore is the fact that alcohols (including rubbing alcohol, perfume, aftershave, and liquor) inactivate the toxins that are found in the nematocysts of jellyfish and other coelenterates. An important fact is that anaphylaxis rarely develops more than 1 hour after exposure. In case of cutaneous exposure to chemicals, "field decontamination ... has been shown to significantly reduce extent of burn." One common sense tip is simply using the open end of the suction tubing if there is a large amount of material to suction from someone's pharynx. Something that most people do not like to discuss is that the rescuer may also vomit when resuscitating someone who has vomited and obstructed his airway. Finally, the authors make abundantly clear that airway obstruction is one of the most common treatable causes of prehospital death.

Under airway and ventilation, the authors state that mouth-to-mouth ventilation should seldom be necessary in the field. ("Seldom" should probably be replaced with "never.") They consider oxygen-powered devices easy to use but also potentially the most dangerous. (Earlier in the text, however, they advocate using such devices to ventilate someone who has been intubated—a contradiction.) A point that is sometimes missed by our students (and generates much discussion during our ACLS course) is that endotracheal intubation should not be used as the initial method of managing the airway during cardiac arrest. Two excellent points made in the section on operational procedures are (1) It is riskier not to treat someone who needs care than to treat someone in good faith with less than full permission (the medical equivalent of "It is better to ask forgiveness than permission") and (2) "The best defense against any legal question of consent, competence, and the need for care is a good medical record." Finally, if the paramedics are treating someone in cardiac arrest in a physician's office and the physician is being helpful, the authors' recommend accepting his help.

There is also judicious use of humor to make points. When using charcoal to treat poisoning, the paramedic is urged to assure that the nasogastric tube is indeed in the stomach because "Charcoal is not helpful to the lungs." When delivering babies, one must remember that "Babies are slippery. It is considered poor form to drop one." and that "Babies have poor temperature regulation and no clothes."

On the other hand, the book contains some suggestions that are fairly well off the beaten path. In the section on multiple patient assessment, for instance, the authors talk about medical command (unfortunately not in the entire context of incident command for a multiple casualty incident, or MCI) and then mention only triage and transport teams (normally called sectors). They omit treatment and staging (supply) sectors at an MCI, all of which must be set up initially even before treatment is initiated. Under cardiac arrest, they suggest first initiating CPR and then checking rhythm with a monitor. This is surprising considering the American Heart Association's (AHA) push for early defibrillation—and that this book is copyright 1996. Their algorithms are somewhat different from AHA recommendations. For instance, they use only one dose of a given antiarrhythmic before moving on to the next drug. They also add lidocaine to the Pulseless Electrical Activity (PEA) algorithm in which the pulse is > 60. During resuscitation from ventricular fibrillation, if defibrillation has been successful at 360 joules and ventricular fibrillation recurs, the authors recommend defibrillation beginning at 200 joules again. The dose of atropine is specified as 1 mg I.V. or 2 mg via endotracheal tube (as opposed to the 0.03-0.04 mg/kg recommended by the AHA). The authors are also advocates for the consideration of sodium bicarbonate after 10 minutes of a full arrest and every 10 minutes after that. (For re-emphasis that ventilation and rapid restoration of perfusion are the mainstays of acid-base regulation during resuscitation and a long list of reasons not to use sodium bicarbonate, see the latest AHA Guidelines for CPR and ECC.[1]) In the case of chest pain, auscultation may reveal "rales, crackles, wheezes..." (I thought crackles and rales were synonyms.) In a narrow complex tachycardia with a regular rhythm and an unconscious patient with a pulse > 150, after an initial cardioversion at 100 joules without "discharge," defibrillation at 100 joules (unsynchronized) is the next step. For ventricular fibrillation in the pediatric patient, the authors specify pediatric paddles; for infants above 10 kg, adult paddles have been shown to provide more effective energy delivery to the myocardium. They give their pediatric doses in mL/kg after (sometimes) specifying drug concentration instead of the less confusing mg/kg.

For face and neck trauma, airway control is effected by chin lift, manual traction, or towel clip to the tongue (Ouch!). For air embolism, the authors emphasize looking for a pneumothorax or tension pneumothorax. They also recommend using a *decompression chamber* to treat decompression sickness. For respiratory arrest, one should use pocket mask or bag-valve-mask (BVM) for ventilation. Current AHA recommendations are for two people if BVM ventilation is used on the nonintubated patient. It has been shown that rescuers are unable

to deliver even the minimum recommended tidal volume if they are using a BVM without assistance of another person.[2] The authors make the blanket statement that the head tilt/neck lift method of opening the airway cannot be simulated on mannequins. While this may be true on some, it cannot be universally applied. A diagram purportedly showing proper technique for BVM ventilation shows the rescuer with four fingers under the jaw and only the thumb holding the mask on the victim's face. In the airway maintenance section there is a statement that one should not hear breath sounds over the stomach. They can be referred there from the lungs; one should not hear gurgling (the "toilet flush sound") over the stomach. For the victim with upper airway trauma, the authors opine that intubation should not be aggressively pursued; in the Advanced Trauma Life Support (ATLS) course, however, one will fail the "stab wound to the neck" scenario if your first action is not oral endotracheal intubation. The authors explain that pulse oximetry uses the differences in color between hemoglobin and oxyhemoglobin to give a reading. (Perhaps this is being overly critical, but oversimplification is not always a virtue.) A somewhat anachronistic view is presented that "All I.V.s started in the field are considered contaminated and must be changed within 6 hours of insertion." Field technique for I.V. insertion parallels that used in hospitals, primarily because clinical practice for I.V. insertion takes place in hospitals. For spinal splinting, the authors suggest having an assistant apply gentle traction along the axis of the spine; this was changed several years ago to just stabilization. Likewise, the use of sandbags for maintaining spinal stabilization is presently eschewed in favor of foam blocks. The authors make the surprising statement that tracheal shift is rarely present in the case of a tension pneumothorax. Last, they are overly generous with their estimates of F_{IO_2} of various devices, including 0.90 from a non-rebreather with an oxygen flowrate of 10 L/min and 0.8 and 1.0 from mouth-to-mask with oxygen flows respectively of 15 and 30 L/min.

In the big picture, the errors and strange suggestions are few in number, relatively benign in effect, and should not confuse the practicing paramedic. (And if they do confuse, perhaps the paramedic should not be practicing—at least on patients!) Overall, this handbook has much to offer the field paramedic. It could be a useful addition to one's library (although its size precludes its being carried in the pocket).

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Taber's Cyclopedic Medical Dictionary, 18th Edition, edited by CL Thomas MD. Hardcover, 2,439 pages, illustrated. Philadelphia: FA Davis, 1997. $32.95.

This new edition of **Taber's Cyclopedic Medical Dictionary**, while not a pocket-sized book, is a convenient size—lightweight and easy to carry in a backpack or briefcase. The dictionary comes with thumb tabs for $32.95, without thumb tabs for $29.95, or in a more costly ($69) deluxe leather-bound, gild-edged edition (a nice gift idea for the graduating health professional). The editors of the lexicon have incorporated several full-color illustrations (377, to be exact) and 2-color line drawings (approximately 180) that emphasize some of the definitions. In addition, the editors have added several useful appendices.

It is difficult for this reviewer to be completely convinced of this lexicon's value to the respiratory care practitioner (RCP) because "cyclopedic"—meaning exhaustive, broad, and varied—seems to apply more to nursing than to any of the other health professions. Nursing implications are listed with many of the definitions; and 3 of the 21 appendices are devoted to nursing diagnostic codes, conceptual models, and organizations. While I acknowledge the nursing responsibilities associated with many respiratory care procedures, omitting the respiratory care professional's role as well as those of other health professions narrows the focus of a 'cyclopedic' work. The roles of dieticians, physician's assistants, physical and occupational therapists, emergency medical technicians, and others, although listed as professional designations in an appendix, are either ill defined or undefined. (I am astonished that the American Association for Respiratory Care is left out of the appendix, Health Care Resource Organizations, and that the only professional associations listed were those for nursing.)

With that aside, I am interested in the treatment of respiratory terminology in the body of the lexicon. BiPAP is defined as "bilevel continuous positive airway pressure." I believe that Respironics Inc would agree that BiPAP is a trade name. F_{IO_2} is simply defined as "fractional concentration of oxygen." For most RCPs the term means the fractional concentration of *inspired* oxygen. F_{DO_2} (fractional concentration of *delivered* oxygen), a term used in RESPIRATORY CARE, is not explained. P_{aCO_2}, P_{aO_2}, and S_{aO_2} are listed satisfactorily. The definitions for *tracheostomy* and *tracheotomy* do little to alleviate the confusion of these terms. (For editorial purposes at RESPIRATORY CARE, we use *tracheostomy* to mean the opening, or the stoma, and *tracheotomy* to mean the surgical procedure.) **Taber's** chooses to define these words as synonyms.

An error occurs on Page 1,663; the waveform representing Cheyne-Stokes breathing is missing—the grid and apnea mark are present.

On Page 1,134, the editors use a 4-color representation of the lung with a set-out line drawing depicting the arteriole, pulmonary capillaries, alveolus, venule, and alveolar duct. The line drawing seems accurate and enhances the definition.

If one can put aside the emphasis on nursing, the dictionary is helpful in an elementary way. Overall, the use of 2- and 4-color photographs and line drawings and the appendices covering Universal Precautions, Diagnostic Codes, Anatomy, and Units of Measurement make it worth the nominal cost. The appendix on computer terminology, although probably soon to be outdated, may be of use to the novice health care computer user.

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Letters

A Reminder of Risks

I congratulate the authors of the paper "CO_2 Retention Secondary to Laparoscopic Cholecystectomy" published in the February issue of the Journal.[1] The article is informative and provides a valuable learning opportunity. I am surprised that there has not been more study done on the increased risk of hypercarbia secondary to laparoscopic procedures. I found the blood gas results obtained while the patient was in the surgical intensive care unit amazing. To find a patient with severe acidosis after a laparoscopic procedure astonished me. While reading this article, I was reminded that all surgical procedures have risks. I thank the authors for reminding us that laparoscopic procedures do have risks that we as respiratory care practitioners must remember to monitor.

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Central Venous Cannulation—When the Risks Outweigh the Benefits

I read with interest Dr Sinkovič's case report "Acute Respiratory Failure from Neck Hematoma Following Venous Cannulation in a Patient Receiving Streptokinase,"[1] but I respectfully disagree with Dr Sinkovič's management. When one performs an invasive procedure on a critically ill patient, extra caution should be applied. Repeated attempts to cannulate the internal jugular vein (IJVC) may result in hematoma formation at the site, particularly in patients with disorders of hemostasis or in those receiving anticoagulation therapy. However, in the case reported by Dr Sinkovič, IJVC was the site of choice despite several failed attempts in an anticoagulated patient. Manual compression should have been applied immediately with the first sign of swelling at the puncture site, rather than simply observing the patient for 1 hour, when a large hematoma had developed that resulted in intubation and mechanical ventilation for treatment of respiratory failure, discontinuation of the intravenous heparin, and even the need to reverse the anticoagulated state by administering fresh frozen plasma.

In view of the recent acute myocardial infarction in this patient, the IJVC should have never been attempted because of the potential serial sequels—hematoma and discontinuation of anticoagulation therapy. A femoral vein cannulation (FVC) should have been attempted instead. An FVC would have allowed continuation of heparin while the benefits of streptokinase to salvage the myocardium were continued. The advantages of FVC over IJVC are that hemorrhage is more easily controlled in FVC and compression of the airways or the carotid(s) and consequent hypoxia may be avoided. The main complications of FVC are deep vein thrombosis and infection—both of which can usually be avoided.

Ultrasound-guided IJVC in patients who are not receiving thrombolytic therapy carries a 1.7% rate of carotid artery puncture and a 0.2% rate of hematoma formation.[2] FVC in critically ill patients offers an alternative site of insertion and has been proven to have an acceptably low rate of clinically important complications.[3,4]

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Dr Sinkovič responds:

I appreciate Dr Alkhuja's letter concerning my case report,[1] reflecting his interests in this special field of intensive care medicine.

In my patient, ventricular arrhythmias (Lown's Class II, III, IVB) and bradycardia (40/min) with widened QRS complexes, appearing 4 hours after streptokinase therapy had been completed, had to be treated by intravenous medications (lidocaine and atropine), but, unluckily, the peripheral intravenous line was no longer patent.[1] In acute myocardial infarction, widened QRS complexes with bradycardia usually require short-term cardiac pacing because these findings are often followed by a slow ventricular-escape rhythm due to complete atrioventricular block.[2] During thrombolytic therapy, transcutaneous external cardiac pacing, if available, is particularly useful to treat bradyarrhythmias if they do not respond to atropine. In our ICU at the time of the patient's presentation, transcutaneous cardiac pacing was not yet available, and transvenous cardiac pacing would have been necessary if medical treatment failed. Although the most prone to manual external compression, the simplest, most direct route to the right ventricle would be provided by the intravenous route and would allow emergency cardiac pacing in time. Full-dose systemic anticoagulation and thrombolytic therapy are only relative contraindications to routine intravenous cannulation under such circumstances.[3]

In my patient, swelling of the neck after hematoma formation was noticed 3 hours after I.V. cannulation had been performed, indicating slow and continuous bleeding from eroded vessels in the neck area. I agree with Dr Alkhuja's statement that manual compression at the puncture site is important because it is effective in preventing hematoma formation, at least as long as it is continued. In my patient, thrombolytic

therapy had been completed several hours previously but as soon as manual compression was stopped, bleeding started again because a protective hemostatic plug could not be formed on the injured vessel wall. The main constituent of the coagulation system (ie, fibrinogen) but also plasminogen and inhibitors of fibrinolysis were consumed by pharmacologically induced fibrinolysis and only reversal of the acquired coagulopathy by infusion of plasma proteins could stop the bleeding. Waiting for spontaneous recovery of native fibrinogen, normal fibrin in the clot, and thrombin-coagulable proteins would take another 10-15 hours because considerable recovery is achieved only 25 hours after the start of streptokinase therapy.[4]

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January Issue—A Useful Clinical Tool

My instructors—in helping me prepare to become Registry eligible—have relentlessly stressed the importance of total patient assessment. It is encouraging to see that concept brought to national attention in the January 1997 issue of the Journal. The article, "The Respiratory Care Practitioner in Extended Care Facilities" by Kevin A Cornish RRT[1] stresses the importance of preparation of the respiratory practitioner to provide diversified care.

As a CRTT, I have practiced in extended care facilities, also known as subacute care facilities, and understand the points Mr Cornish made. In the article, he discusses the link between the care provided by respiratory care practitioners in these facilities and the continuum of total patient care.

Extended care facilities promote individualized assessment, critical evaluation underlined by education and knowledge, to best care for the patient. Clinical evaluation and communication with other practitioners (physicians, nurses, and rehabilitation teams) are a vital part of providing care that minimizes future hospital admissions. I think it is important to remember that knowledge and understanding of the different levels of care required by patients provides consistency to the individual patient.

I commend the educators who stress the importance of critical evaluation of the patient, support the concept of a broad scope of practice, and emphasize the importance of our profession at all levels of care.

I also commend Mr Cornish for his emphasis on the importance of extended care facilities in the continuum of care and Respiratory Care Journal for the entirety of the January 1997 issue. The issue should be regarded as a necessary clinical tool for students, offering encouragement in a "not so encouraging time" in health care.

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A Tribute to Jack Emerson

My career as an inhalation therapist began August 1, 1968. Although our department had a few MA-1s, I first applied mechanical ventilation with an Emerson ventilator. A few years later when I cared for a child with myasthenia gravis, my choice of ventilators was the Emerson. Jack Emerson heard of our endeavor and brought a pediatric piston for our patient, Stevie, to Grady Memorial Hospital in Atlanta. He stayed on for a few days to make sure that our patient did well.

For years after that, I would run into him at AAIT/AART/AARC Annual Meetings. He seemed to know everyone. I also put to good use some of his inventions (PEEP and demand valves) for many more years.

Today, I heard that Jack Emerson died. What a force he has been. *Breathe a sigh, Jack. You earned it!*

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RETROSPECTROSCOPE

Premature Science and Immature Lungs Part II. Chemical Warfare and the Newly Born

In Part I of "Premature Science and Immature Lungs" (AMERICAN REVIEW OF RESPIRATORY DISEASE, July 1977) I gave an account of the beginnings of the quantitative approach to surface tension (Laplace's law [1806], Bowditch's remarkable translation of it into English, with annotations and corrections [1829-39], and Young's work on cohesion of fluids [1804-05] which antedated Laplace's law) and of the considerable interest of physicists, chemists, and general physiologists in surface tension and interfacial phenomena. However, the discovery of surface forces was premature for mammalian physiologists; 123 years elapsed before Neergaard did his now-famous studies on surface forces generated by alveolar tissue-air interfaces. Neergaard's work was also premature for pulmonary physiologists who came after him, and another 25 years elapsed before his work was rediscovered. Part II tells some highlights of the 30-year post-Neergaard period up to the 1959 paper of Avery and Mead.

"Hyaline Membranes"

Not because of Neergaard's paper, but coincidentally with its publication, pediatricians and obstetricians became increasingly interested in the diagnosis and treatment of atelectasis of the newborn and in the pathologic state of lungs of infants who died with this syndrome. Curiously, they centered their interest on "hyaline (or hyaline-like) membranes" that pathologists, depending on the intensity of their convictions, found in the alveoli of all, most, many, or some newborn infants that had severe respiratory distress at birth and died shortly thereafter. These "membranes" had been described as early as 1903. They were the subject of three papers in the 1920s, nine in the 1930s, thirteen in the 1940s, and many more in the 1950s. In retrospect, they were not really membranes at all (in the sense of being a thin sheet covering or lining a tissue); they were merely a translucent, homogeneous something that stained with eosin. And they were the *result*, not the cause, of severe asphyxia and the consequent process of deterioration of cells, tissues, and the baby. More important, the "membranes" diverted the attention of most investigators away from atelectasis and away from new concepts that might have led to finding the cause of the disorder; in retrospect they were red (eosinophilic) herrings drawn across the trail and distracted the scientific hounds.

The history of science contains many examples of investigators who considered a problem solved because they had named it (or renamed it); it seems that this happened in the case of "hyaline membrane disease."* The history of science also includes more scientists whose goal was to find the *presence* of something rather than the *absence* of something. Thus, in the late nineteenth century, when new pathogenic bacteria were discovered almost monthly and the *presence* of each was associated with a disease, nutritionists had a hard time selling the medical public that the *absence* of specific substances, called vitamins, could also cause disease. Fascination with the *presence* of hyaline membranes may well have delayed the discovery of the *absence* of surfactant.

Not everyone concentrated his attention on hyaline membranes. Wilson and Farber in 1933 (27) emphasized the importance of "cohesion of the moist surfaces in collapsed and airless lungs." This "stickiness," they wrote, offers considerable resistance to the entry of air, and a relatively great force

> ...is required to overcome it and to separate the bronchiolar and alveolar walls during the initial expansion. The first breath of every new-born baby may thus be its most difficult one, and especially vigorous inspiratory efforts must be maintained for a variable period

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*At one time or another, the syndrome was called by 13 different names. The abundance of names and the paucity of evidence linking the name and the cause of the disease is another example of the "law" that the amount of knowledge of the *cause* of a disease is inversely proportional to the square of the *number of names* acquired by the disease.

> after birth until the normal atelectasis is finally overcome. This initial resistance of the atelectatic lungs to expansion is always present and contributes to the maintenance of atelectasis to a pathologic extent....
>
> It is believed that this initially lessened distensibility of the lungs after they have stood for several hours in a state of passive collapse or after they have been entirely exhausted of air by forceful suction is due to the cohesion of the moist surfaces of the air passages, and that this is an important factor in the maintenance of the fetal state of atelectasis after birth; it is a factor that is always present, and when combined with any of the other conditions to be discussed, it causes the state of atelectasis to persist to a pathologic extent.

Wilson and Farber in their report referred neither to Neergaard's research nor to Thomas Young's 1806 paper on cohesion. They noted that lungs that were collapsed and contained neither air nor liquid required considerable force to expand them initially and then less on subsequent breaths. They realized that babies born with atelectasis continued to have it for variable periods after birth but didn't connect it with the continued absence of something in the alveoli (other than the absence of air).

In 1947, Gruenwald (28), also not knowing of Neergaard's work, determined, first using saline and then air, the smallest pressure needed at autopsy to expand nonaerated lungs of stillborn infants and those dying shortly after birth. In 15 infants, the pressure was 9 cm H_2O for filling with saline and 18 for filling with air. This difference he attributed to the existence of surface tension at the tissue-air interface and its absence at tissue-saline interface. He decided to see whether anything could be done to decrease surface tension at the tissue-air interface. He introduced amyl acetate into the lungs by two methods; one consisted of forcing in air containing amyl acetate vapor and the other of rinsing the lung with 0.25 per cent amyl acetate in saline before introducing air. He reported his findings:

> There was a marked reduction in the pressure necessary to introduce air when amyl acetate was used by either of the two methods. The reduction amounts to nearly one-half the difference between the values for pure saline and air. This correlates well with the reduction of surface tension by amyl acetate, as shown roughly by the height of a fluid column in a capillary (33 mm for saline, and 21 mm for amyl acetate solution).
>
> Another change was noted when amyl acetate was used. The aeration was more diffuse than usual, affecting larger portions of a lobe at the same time without immediately expanding them completely. In this respect, too, the condition was intermediate between normal aeration and expansion with saline. It is thus demonstrable that surface tension is a major factor in the resistance of the lung of the newborn to aeration but does not interfere with the aspiration of fluid.

He then concluded by suggesting that the addition of surface active agents to the infant's inspired air or oxygen might be helpful in relieving initial atelectasis of newborn infants. Gruenwald, being a pathologist, could not put his suggestion into action; the pediatricians and obstetricians continued to debate the origin of hyaline membranes (29, 30).

Macklin and the Pneumonocytes

Staub, Clements, Permutt, and Proctor have recently written a brief biography and an appreciation of Macklin's work (31). Macklin spent most of his academic career as Professor of Histology and Embryology at the University of Western Ontario in Canada. Much of it was occupied studying the airways, the pulmonary circulation, and macrophages; some of it was devoted to speculation on surface forces in alveoli. Macklin, like others, was unaware of Neergaard's work and reasoning. Macklin's first reference to surface tension was a brief sentence in 1946 in a long paper describing epicytes (32). In it he stated that epicytes ("residual alveolar epithelial cells") may be in the class of a secretory cell and play a useful role in conditioning the surface fluid film of alveoli. "It may be," he wrote, "that it [the epicyte] contributes something to the circumscribing fluid film of the alveolar capillary wall which is advantageous in external respiration." In this 1946 statement he neither defined the "something" nor explained how it was "advantageous," but obviously he realized that it might be physiologically important, though he didn't specify how.

In 1954, Macklin (33) published his well-known paper on the alveolar mucoid film and the epicytes, which he had now renamed "pneumonocytes." In it he first noted that on September 4, 1953 (at the International Physiological Congress in Montreal) he had elaborated on his 1946 views and had suggested that a watery mucoid film, a hydrated secretion of granular pneumonocytes, provided a noncellular interface between alveolar air and the thin alveolar epithelium. He then elaborated on his 1953 suggestion by making a clear statement of his concept that a thin, aqueous, mucoid, cohesive flexible film, about 0.2 μm thick, covers alveolar walls and, as he put it:

> ...[performs] vital functions, such as assisting in the removal of fine living and dead particulate matter, the maintenance of a constant favourable alveolar surface tension, the facilitation of gaseous exchange, the protection of the underlying tissue from desiccation, and the suppression of invading micro-organisms. Its mechanism of production normally insures a constancy of volume, thickness, water and solid content, and other features.
>
> There is evidence pointing to the granular pneumonocytes as the originators of the secretion which composes this film....
>
> It may be causally related to the myelin figures

> (Leathes 1925) which are seen by phase-contrast microscopy to emerge in great abundance from the surfaces of the alveolar walls in sections of fresh lung mounted in water or in physiological saline solution, for they are generated in hydrophilic material (Frey-Wyssling 1953b). It may have something to do with the inhibition of bubble formation, for in life no air-bubbles are normally formed in the alveoli. If they were so formed, in quantity, the effect would be disastrous.

Staub and associates (31) wrote:

> Although Macklin did not overtly state that the granular pneumonocytes secrete surfactant, it is obvious that he recognized "myelinogens" (phospholipids) in the alveolar lining and knew that they were connected with the osmiophilic lamellar bodies of the granular pneumonocytes. He also recognized the aqueous, mucopolysaccharide-rich nature of the alveolar surface and the need to regulate alveolar surface tension.

Although the "aqueous, mucopolysaccharide-rich" film *is* indeed there, Macklin did not appreciate that on top of it is a monomolecular layer of phospholipid-containing surfactant and that this lowers surface tension of the film when alveoli become smaller. Macklin thought that the function of the film was to maintain a "*constant* favorable alveolar surface tension" (italics added); if he had written "constant*ly* favorable" he would have been correct—but instead he wrote "constant ... tension." And so direct, tangible evidence of the existence of pulmonary surfactant was still a few years away.

The Role of Chemical Warfare Research

Macklin's work on the pneumonocytes was supported by the National Cancer Institute of Canada, but his earlier work on alveolar size was supported by the Canadian Chemical Warfare Laboratories (34, 35). Next in our highlights come the studies of Radford, Pattle, and Clements. Before going on to the research of each, it's worth recording in the history of science that their papers—ones that were crucial to the discovery of surfactant—were supported by the chemical warfare laboratories of the United States and England. Radford worked at Harvard supported by a contract from the Medical Research Laboratories of the U.S. Army Chemical Center; Pattle was a physicist-physiologist working full time for the British Chemical Defence Experimental Establishment at Porton, England, and Clements was a full-time clinical physiologist working at Edgewood, Md., in the U.S. Army Chemical Center. All had the overall mission of preventing, diagnosing, and treating damage to personnel by a variety of war gases, such as the new nerve gases that cause neuromuscular block and respiratory paralysis, and phosgene, which causes pulmonary edema.

During World War II, the U.S. laboratories had an all-star uniformed staff including Alfred Gilman, Oscar Bodansky, George Koelle, Maurice Strauss, Hale Ham, Richard Bing, Clarke Wescoe, Timothy Talbot, Frederick Crescitelli, Frederick Phillips, and James Whittenberger; the staff at Porton included Sir Charles Lovatt Evans (physiology), J.H. Gaddum (pharmacology), and G.R. (later Sir Roy) Cameron (pathology). When peace came, medical research to combat the effects of chemical warfare continued on both sides of the Atlantic, both intramurally in government laboratories and extramurally supported by contracts at universities.

Chemical warfare has never been a popular wartime weapon and, except for isolated instances, has not been used since World War I. In a sense then, the input of dollars and manpower and the output of research for chemical defense have not been applied to the military needs for which they were intended. But somehow or other, though never planned that way by any general staff or commission or task force, research supported by chemical warfare dollars led to our first real understanding of the nature and cause of the respiratory distress syndrome in the newborn; that, in turn, proved essential for the first effective management of a disorder that, until a few years ago, caused the death of at least 20,000 newborn babies each year in this country alone. If you had known this when the Mansfield amendment (which prohibited Department of Defense scientists from doing basic research) was being debated by Congress, you might have testified that a lot of good science can come out of "military" research when the right commanding officer, the right scientific director, and the right scientists come together at the right moment. Basic research on alveolar surfactant never won any military battles, but it largely won the war against atelectasis of the newborn.

So much for this aside. Now for the work of Radford.

Radford and Alveolar Surface Area

At last we come to someone writing in English who knew of Neergaard's work.[7] Radford, before receiving his M.D. degree at Harvard (1946), went to Massachusetts Institute of Technology and had an excellent background in the physical sciences when he joined the physiology department first at Harvard Medical School (1949-52) and then at Harvard's School of Public Health (1952-55). Not only that but he had an unusual dual knack of knowing how to apply physical knowledge to physiology and medicine and how to teach: when I organized a Medical Faculty Training Program at the University of Pennsylvania in 1956-57, designed to acquaint or

[7] Christie and McIntosh (1934) list Neergaard and Wirz (1927) and Neergaard (1929) in their bibliography but refer to the 1929 paper in their text only once, and incorrectly, in a listing of previous workers who had measured intrapleural pressure: Neergaard in 1929 reported only his work on excised lungs, distended either with air or fluid.

reacquaint faculty members with both basic sciences and basic medical sciences, I turned to Radford (then at DuPont's Haskell Research Laboratories in Delaware) to give a course on "The Physical Basis for Analysis of Biological Processes" for the group, and a splendid course it was.

Radford didn't set out in his research to find the cause and treatment for atelectasis of the newborn. What he wanted to do in the early 1950s was to estimate the total surface area of the alveoli of mammalian lungs. The Harvard group of Whittenberger, Mead, and associates, at the School of Public Health, had a deep interest in pulmonary physiology, and alveolar surface area was an important number to those interested in diffusion and gas exchange between alveoli and pulmonary capillary blood. Radford believed that the estimates of this area that had been made using histologic methods (on lungs scarcely in a physiologic state) tended to result in overestimates of the number of alveoli and of the total alveolar surface. So he decided to use a physical method based on surface tension and energy. His work, which began in 1952, was published in 1954 (his first published paper) (36). In it he wrote:

> The method of estimating lung surface described in this report is based on physical measurements of the lungs and avoids any assumption of size or configuration of respiratory structures. The calculation depends on consideration of the changes in free energy of the lungs as they are deflated. During inflation, energy is stored by deforming elastic elements, and by the creation of a large air-liquid interface. If the lungs are deflated by allowing volume equilibrium at successive pressure decrements, the stored energy released during deflation can be measured....
>
> As will be shown below, it is possible to estimate the contribution of elastic energy to the total free energy and obtain the surface free energy by difference....
>
> Gas-free animal lungs are prepared by allowing the pulmonary blood flow to absorb the alveolar gases, after initially hyperventilating the lungs with pure oxygen. These completely deflated lungs are removed, filled with isotonic NaCl solution while suspended in a saline bath, and the "elastic" curve is obtained during deflation by allowing the volume to reach equilibrium at each pressure. This curve is then compared with the deflation curve subsequently obtained in the same way after air inflation at the same temperature.

Here is another example of the Lost and Found Phenomenon (37). Radford did his initial experiments completely independently of Neergaard's work and did not know of Neergaard's paper until later in his experiments, when Mead found it in searching the literature. However, Radford's rediscovery provided a necessary confirmation of Neergaard's conclusions on the importance of surface tension in alveolar expansion and retraction. Radford then set out to calculate alveolar surface area. Using a figure of 50 dynes per cm (that of human serum) as that of the interfacial layer between tissue fluid and air in alveoli, he calculated that the total alveolar surface area was about one-tenth that calculated by those who had used histologic methods. He then commented on the difference in numbers obtained by the new and the old methods:

> The striking difference between the estimate for human lungs of 5 to 10 m^2 by the method discussed in this report and the estimates of 50 to 100 m^2 obtained from histologic measurements, deserves additional comment. None of the assumptions on which the surface energy method depends could account for this difference unless the lung surface were a semi-solid phase or consisted of a highly surface-active substance. *Neither of these possibilities is at all likely* [italics added].

Radford had come to a fork in the road and had to decide which fork to take: one, that his theory, assumptions, and calculations were correct, or the other, that his assumed value for surface tension was wrong and that instead, the "lung surface consisted of a highly surface-active substance." He chose the former, and missed the opportunity to be the first to find and identify pulmonary surfactant.

We might speculate why Radford had more faith in his values than in the estimates of histologists. In the 1950s and earlier, anatomists did not concern themselves with studying lungs under physiologic conditions and their data were suspect on this account. Histologists who studied lungs closer to their physiological state got smaller values for alveolar area; others got the higher figures. It was logical for Radford to question the histologists' data; it would have been illogical in 1954 to have assumed the existence of a biologic substance that could lower surface tension to extremely low values. Radford therefore accepted a value of 50 dynes per cm for the surface tension of the alveolar film. Why wasn't he influenced more strongly by Neergaard's measurements of surface tension? Radford said in his paper (36):

> Neergaard ... measured the surface tension coefficient of lung tissue extracts, using the method of Lenard *et al.* His results varied from 35 to 41 dynes/cm; in view of errors of the method and the presence of intracellular substances, these values cannot be applied to intact lungs. von Neergaard found no conclusive evidence for a surface-active film in the lung, and concluded that the interfacial layer is probably an aqueous solution having a surface tension coefficient near that of serum.

There were no published translations of Neergaard's German into English in 1954. Now there are two (38 a, b). The translators agree that what Neergaard wrote was as follows:

> In such extracts [aqueous extracts obtained from the lung parenchyma] ... I found consistently unexpectedly low values of 35-41 dynes. From a physic-

> ochemical point of view, it would be understandable that surface-active substances gradually accumulate at the alveolar surface.... The uncertainty about our results, arising from the fact that we do not know the exact surface tension in the alveoli, cannot therefore be fundamental, because the surface tension of the gum solution is very low, and that in the alveoli must also be very low, that is, at the lower biological limit. ...the error cannot be great; it is probably less than individual and species differences. Our main findings and their order of magnitude remain unchanged; nevertheless, the question should be further investigated.

I interpret Neergaard's words (in these translations) as an expression of his conviction that the surface tension at the alveolar-air interface must be very low, but that scientific caution compelled him to call for further investigation. Radford may have interpreted Neergaard's words as an expression of uncertainty about his method and that the surface tension must be near that of plasma. Further, Radford knew that lung tissue contained lipids that mixed with real lung tissue (alveolar walls), could give a misleadingly low value. Actually to a "fat and soap" scientist of Neergaard's time (and Neergaard consulted with Professor Scherrer of Zurich Federal Technological College who was quite sophisticated in surface chemistry), the difference between 50 dynes per cm (the value for plasma) and 35 (Neergaard's lowest value for lung extracts) was enormous; as Neergaard put it, 35 was "at the lower biological limit." However, Radford may have interpreted this difference as being within experimental error. We now know that pulmonary surfactant is unique in lowering surface tension far below 35 dynes per cm, to 10 dynes per cm or less.

Sometimes, an incorrect assumption or conclusion holds back scientific advance. In this case, Radford's did not, for it served as an important stimulus to others, including Clements a few years later, to reopen the issue. More of Clements soon; but first we should discuss Pattle and his noncollapsing foam.

Pattle and Pulmonary Edema Foam

Pattle became interested in science at an early age, "six or possibly earlier ..., particularly chemicals and such things as frogs and beetles." He went to Westminster School at the age of 14, started with the usual classical curriculum (which he didn't think did any harm), but switched to science a year later. He read the literature thoroughly for a prize essay on surface tension; he didn't win the prize because he never wrote the essay, but he acquired a good knowledge of surface forces at an early age.

Pattle received first class honours in physics at Oxford in 1940, then worked on medical aspects of the war effort from 1941 to 1943 with the British neurophysiologist, Weddell; his research dealt largely with clinical electromyography, denervated muscle and human pain fibers. In 1943, he joined the Royal Electrical and Mechanical Engineers, concerned mainly with radar, becoming a Captain in 1946. In 1947, while in Germany, he saw an advertisement for biophysicists, and applied and received a post, now in civil service, at the Chemical Defence Experimental Laboratory at Porton, England.

He worked in the Physics Department under H.L. Green and W.R. Lane, noted authorities on particulate clouds, dust, smokes, and mists, and they assigned him the task of studying surface tension in certain liquids intended to be used as aerosols. In the course of this work, he devised a simple method of producing a "raft" of equal-sized bubbles on the surface of a liquid. This "raft" could be measured and manipulated to yield useful information on the properties of the liquids. In 1950, he published a note on mutually immiscible layers, and two full papers, one on the production of small bubbles and the other on the mechanism of action of anti-foams. Though no one then knew it, this was the best possible groundwork for his later, completely unrelated, research.

However, in 1952, after several years of administrative assignments in London, Pattle returned to Porton to work under Dr. Kenneth Wilson in Porton's Physiology Department. (Wilson is now at the U.S. Army Chemical Center in Edgewood and Assistant Professor of Anesthesia at Johns Hopkins.) No one at Porton was then thinking about surface tension in pulmonary alveoli. From November 1952 to July 1953, John Clements joined Wilson's group as an exchange scientist from the U.S. Army Chemical Center—to do collaborative work on the treatment of nerve gas casualties. Johns and Cooper at Edgewood had just introduced mask-to-mask resuscitation for treating patients with respiratory paralysis, and the group at Porton frequently discussed pulmonary compliance and factors affecting it. Clements brought word of Radford's work-in-progress at Harvard and of the influence of surface tension on lung compliance; Neergaard's name had now reached England via Boston and Edgewood.

At this time, Pattle was still interested more in improving preamplifiers and electrodes for electromyography than in pulmonary physiology.

Bubbles continued to intrude into his work. As he put it, "the curse of these microelectrodes was that small bubbles of air kept getting into them, and the only thing you could do was to wait until the bubble had disappeared under the influence of surface tension. This gave me the idea of measuring surface tension by the rate of contraction of a bubble." He became involved in pulmonary research in 1953—not because he wanted to find the cause and treatment of respiratory distress in newborn babies, but because of practical problems, again associated with bubbles. Phosgene kills by producing pulmonary edema so severe that foam fills alveoli and airways and causes asphyxia. One afternoon, Wilson was talking to Pattle over tea when the "Chief," Harry Cullumbine, and Professor Cameron dropped in. Cameron had been full-time at Porton during World War II and became so interested in pulmonary edema that he continued working part time at Porton for years after the end of the war. Cameron was studying experimen-

tally-produced pulmonary edema in goats and losing most of them when foam welled into the airways. Cullumbine had learned of the work of Curry and Nickerson (39), who claimed that antifoam agents were effective in breaking up the foam of pulmonary edema, and Cameron remarked, "Pattle, I hear that you're an expert on foams." Pattle averred that he knew something about them. So Cameron continued, "Can you do something to break the foam in my goats?" Pattle thought he could, and started by repeating the work of Curry and Nickerson. However, when he added several powerful antifoams to a sample of tracheal foam from a rabbit suffering from pulmonary edema, the foam remained unaffected (40). Pattle knew then that the air bubbles of foam originating in alveoli must be covered with the lining layer of the alveolar surface, a unique material that conferred great stability on the bubbles.

Though he concluded that using silicone antifoams was an ineffective way of treatment of phosgene-induced edema, he became intensely interested in stable bubbles. In 1955, he published a note in *Nature* (41); it occupied only a little more than one column, but he packed a lot into it. Here are some excerpts from it:

> In acute lung oedema in the rabbit, fluid and foam are found in the trachea. This foam has an altogether peculiar property, in that it is unaffected by silicone anti-foams; these rapidly destroy the foams produced by shaking oedema fluid or blood serum with air.... Similar foams are obtained from healthy lung by cutting and squeezing under water, or after introduction of saline into the trachea. The stability of such foams is due to an insoluble surface layer on the bubbles; this layer can be attacked by pancreatin or by trypsin.
>
> Oedema foam is thus not produced by agitation of the oedema fluid with air during respiration; it can only have been formed by air originally contained in the fine air spaces of the lung being broken up into bubbles and afterwards expelled into the bronchi and trachea. The lining layer of the bubbles cannot have come from the oedema fluid, and must therefore have formed the original lining layer of the fine air spaces....
>
> In air-saturated water, bubbles (say 40 μ in diameter) from foam from the lung usually remain unchanged in size for long periods (60 min.), whereas similar bubbles from serum and other foams, owing to their internal excess pressure, dissolve and disappear within a few minutes. The surface tension of the lung bubbles is therefore zero; or, to put this another way, the surface pressure of the layer is equal to the surface tension of the underlying liquid....
>
> 40-μ bubbles squeezed from a fragment of the artificially inflated lung of a mature foetal rabbit (within a few seconds of inflation) and kept in air-saturated saline usually contract by about 30 per cent in diameter and then remain stable. The layer is therefore initially formed by rapid surface adsorption from a substance already present in the foetal lung. It probably matures somewhat during the first few hours after birth....
>
> By none of the many methods tried have small bubbles stable in air-saturated saline been regularly produced from blood or from amniotic fluid. The layer is thus not formed from either of these liquids, or from a transudate from the blood. Such bubbles have been formed from nasal (but not, so far, from tracheal) mucus; they are easily formed from a solution of an extract of hog gastric mucosa ('gastric mucin'), and from this solution also a foam resistant to silicone anti-foams has been prepared. These findings suggest that a layer of some form of mucus [Macklin in 1954 had referred to it as a mucoid film], secreted in the depths of the lung, is the source of the insoluble alveolar lining layer.

Pattle also proposed that a very low surface tension was essential if an alveolar lining layer was to prevent continuous transudation into alveoli and consequent pulmonary edema. He wrote:

> Published calculations on the pressure balance between the lung capillaries and the alveoli have disregarded the surface tension at the sharply curved, and probably moist, alveolar wall. If the surface tension were that of an ordinary liquid, enough suction would be exerted to fill the alveoli with a transudate from the capillaries. Means for keeping the surface tension low must therefore be part of the design of the lung. It is thus evident that the alveoli are lined with an insoluble protein layer which can abolish the tension of the alveolar surface.

This concept has not yet been susceptible to direct experimental proof.

Because the Porton laboratories became involved in a study of atmospheric pollutants (as a result of the Great London Smog of December 1952, in which 4,000 people died), Pattle published no more on the subject until 1958 when a full paper, with the same title as the 1955 paper, appeared in the Proceedings of the Royal Society of London (42). This contained a paragraph well worth adding to the above excerpts:

> The finding that the lung lining substance appears only late in the foetal life of the guinea-pig suggests that absence of the lining substance may sometimes be one of the difficulties with which a premature baby has to contend; such a defect may possibly play a part in causing some cases of atelectasis neonatorum. The appearance of a hyaline membrane might possibly be due either to a defective lining layer causing transudation from the blood or to excessive secretion of lining substance. These matters need experimental investigation.

In the meantime, Clements, working in the Chemical Warfare Medical Research Laboratories on the other side of the Atlantic (at Edgewood, Maryland) began to worry seriously about Radford's data and Pattle's statement on zero surface tension.

Clements and Quantitative Studies of Surfactant

Clements, very much like Pattle, decided on his career at an early age. Clements decided to be a scientist when 8 or 9 years old and announced the decision to the world (or at least to his neighborhood) by cutting the letters S-C-I-E-N-T-I-S-T out of the front of a heavy cardboard box, then lining the inside of the box with red paper, installing a flashing light behind the letters and finally placing the completed sign in the front window of the family's house, advertising his wares (figure 1). (I hope that the neighbors got close enough to read SCIENTIST instead of standing off and recognizing only a red light.) He didn't know what kind of scientist he wanted to be and debated a bit between chemical engineering and medicine. His father helped him to decide by sending him on a tour of industrial chemical engineering plants; that tour settled him on medicine.

Fig. 1. A Madison Avenue concept of Clements' window ad.

His decision to go into medical research was reinforced by the state of medical knowledge when he was a medical student between 1943-1947. He found so much in each patient that was unknown that he felt it impossible to have a satisfying career in the practice of medicine. Therefore, after receiving his M.D. in 1947, he skipped the internship and went directly into the department of physiology at Cornell Medical College, headed by Eugene DuBois. Clements thought that he would like to study neurochemistry, especially the intermediary metabolism of brain tissue. But two years later, James Forrestal, Secretary of Defense, issued an appeal to physicians to volunteer for Army service. It is said that his nationwide appeal netted three volunteers, one of whom was Clements, who had been in the Army Specialized Training Program while in medical school.

Fortunately for science (although not for Clements' later income), Clements, having no internship and no license to practice, was assigned for two years to the Army Chemical Corps Medical Research Laboratories at Edgewood, Maryland. At the end of this hitch, Captain Clements decided that he liked the scientific environment at Edgewood and the freedom that he had there to follow his own ideas,** and he stayed on as a scientist in civil service. Edgewood did have some unique aspects: the Research Laboratory's military commander was Colonel John Wood, a highly respected and beloved man, and its civilian Director of Research was D. Bruce Dill, one of America's leading physiologists. And Edgewood was close to Baltimore and Hopkins (where Clements had a part-time appointment as Lecturer in Anesthesiology) and to Philadelphia and Penn (where he was a Research Associate in Physiology).

Clements' first years at Edgewood were spent working on a continuous method for measuring oxygen tension in flowing blood, studying nerve-gas poisoning and methods of treating it (including artificial respiration) and measuring respiratory dead space, airway and pulmonary tissue resistance, and lung compliance.

When Radford submitted the report on his research to Edgewood (1953), Clements thought a great deal about it, and became increasingly unhappy with Radford's conclusion that the histologic estimates of lung area could be so bad as to be ten times too high. Clements applied Laplace's equation to the data and found that calculated surface tension was not 50 dynes per cm at low volumes but fell to values far lower than that, and changed with changing lung volume.

At this point, Elwyn Brown, an anesthesiologist, received a "Greetings" letter and came to Edgewood for a 2-year hitch to work in Clements' lab. At Clements' suggestion, he repeated and extended Radford's work, but he used 50 dynes per cm as the upper limit for surface tension (maximal inspiration) and a calculated value of 15-20 dynes per cm at maximal expiration. Calculated alveolar area now agreed well with histologic estimates of surface area (43). Radford visited Clements and Brown at Edgewood in 1955 and discussed ongoing work on surface tension; he became convinced at this time that lung surface tension was not necessarily fixed at 50 dynes per cm (that of plasma) and that the values he had assumed in 1954 to be correct were not.

Clements, impressed by Pattle's observations on pulmonary edema foam and stable bubbles but unhappy with the fact that they were qualitative estimates, decided the time had come to be quantitative about pulmonary surface tension. Of crucial importance was Clements' decision *not* to use methods that provided a single static value of surface tension (especially of stretched film, as Neergaard had done). Instead he used a dynamic method that provided continuous measure-

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**That he was not completely free from administrative work is attested to by this letter:

Dear Doctor Comroe:
In order to comply with current Army Regulations and directives from the Office of Chief Chemical Officer re proposed publications, manuscripts of scientific papers pertaining to research performed under contractural agreement with these Laboratories must be forwarded in sextuplicate to these Laboratories for clearance prior to submission to the publisher. Present experience indicates that about four weeks are required for action on clearance.
Your cooperation in this matter is appreciated.

Very truly yours.

14 July 1952

John A. Clements
Contract-Project Officer

ments while he altered the surface area in which molecules of surfactant were contained; this permitted him to measure surface tension in a compressed film as well. He constructed a modified surface balance, combining features of both Wilhelmy's balance and the Langmuir-Adam film balance. He was now able to mince whole lungs, place extracts in the balance, and measure continuously the surface tension while alternately compressing and expanding the surface layer. At the 1956 fall meetings of the American Physiological Society, he reported his remarkable findings (44):

> The force-area behavior of surface films prepared from rat, cat and dog lungs has been studied. Surface tension falls from 46-44 to a limiting value of 10-1 dynes per centimeter upon compression of the surface to 30% of its area, with a minimum compressibility of 0.015 to 0.020 cm/dyne. Upon extension the surface shows extreme hysteresis, 10% expansion returning the tension to the upper limit of 46.

In 1957, he published his results more fully (45). Figure 1 from his 1957 paper (see figure 2) shows the close correspondence between the surface tension of plasma and that of lung extract when the surfaces on the Wilhelmy balance were expanded and the marked deviation when they were compressed. Here indeed was a unique surface active material in that it had a variable effect on surface tension such that surface tension decreased to as little as 10 dynes per cm when the surface film was compressed. This obviously was the antiatelectasis factor that was necessary for alveoli to remain open during complete expiration. A few others had toyed with the idea previously, but here was proof. The flood gates were now open for all types of applications to physiology and medicine.

Before going on to further developments, I want to make two comments. The first is that Neergaard's work went unappreciated for 25 years (was "premature") because recognition of its significance required that it come to the attention of scientists who were both trained in physics and mathematics and interested in the lungs. Radford was well trained in physics and Pattle was a physicist. Clements had no mathematics in college and no special training in physics, but a friend taught him rudiments of calculus when both were medical students together and Clements took advanced courses in math at Columbia's night school as soon as he became a physiologist by day at Cornell; physics and physical chemistry, he taught himself. The second point relates to the importance of good communication among scientists. Afternoon tea in British laboratories is not merely a social rite but a way of getting scientists together who, without tea, might never have known of the others' existence. In addition, the close ties between Edgewood, Porton, and Harvard scientists also speeded the exchange of information and ideas. You may read of highly secretive, competitive scientists who clutch their data close to their breasts until their report is published, but

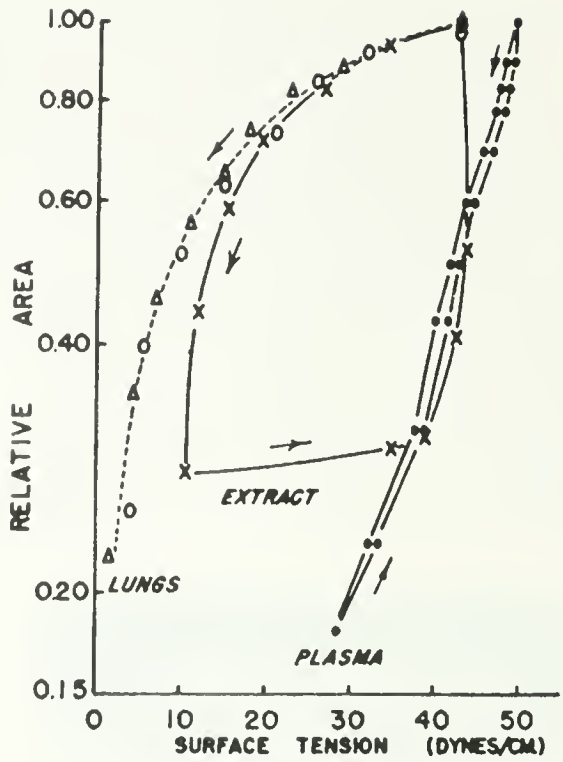

Fig. 2. Variation of surface tension with surface area Upper curve (lungs) calculated on the basis of relative area from Brown's data. Large loop constructed from measurements on lung extract in Wilhelmy balance. Narrow loop constructed from measurements on blood plasma in Wilhelmy balance. (Clements, figure 1, 1957)

Radford, Clements, Pattle, Avery, and Mead were not of this type; each knew what the other was doing long before formal publication. There were no lags here that could be attributed to poor communication.

Avery; Lungs without Surfactant

When Clements constructed his first surface balance in 1955 from sealing wax, chewing gum, string, and other odds and ends, it was probably the only such balance in use in biomedical laboratories. Today, to keep up with the demands of researchers on surface tension, surface balances are made commercially, not by one mechanic working in his garage on weekends, but by at least seven widely known instrument companies. The boom began when Avery and Mead proved that something was wrong with pulmonary surfactant in babies born with respiratory distress syndrome—i.e., what was wrong was that it wasn't there.

Mary Ellen Avery received her M.D. degree from Johns Hopkins in 1952. Within a few months, she received a discouraging blow—she learned that she had pulmonary tuberculosis, and she was shipped off to Trudeau Sanitarium. When she got there and was labeled "complete bed rest" (chemother-

apy of tuberculosis was just in its infancy) with the prospect of two years in bed, she began to do what every good scientist does—she doubted the rationale of established regimens. Why, she thought, does lying on one's back for two years have any beneficial effect on the course of pulmonary tuberculosis? Not getting any good answer, she checked out of Trudeau after two days, went home for a year, then abroad for six months and then started her internship at Hopkins. But the experience was probably critical in her choice of career. One can't be laid up with pulmonary disease for a year or more without thinking and reading and then thinking some more about lungs, and so she became a pediatrician with a subspecialty in pulmonary disease.

During her residency in pediatrics, over and over again she watched newborn babies either taking their first breath or not taking it and she couldn't get answers to penetrating questions about why *yes* and why *no*. Worse yet, she saw clinicians treating respiratory failure in the newborn with the Bloxsom apparatus which at best produced two breaths per minute. So she decided that absolutely essential to good diagnosis and care of the newborn was *new knowledge* (to replace hallowed customs) and if she was ever to contribute some herself, she needed advanced training in research. She consulted Richard Riley at Hopkins and he suggested several centers. She received a two-year special traineeship from the N.I.H. and chose the Boston Lying-in Hospital because Clement Smith was there, making physiological measurements of respiration in newborn babies, and Whittenberger and Mead worked across the street in the School of Public Health. Thus she had the best of both worlds: she could work with the physiologists between 9 and 5 and go across the street to observe the newly born in the early mornings and evenings.

Mead was interested in studying pulmonary edema in dogs (again supported by a contract from the Army Chemical Center) and he asked her to do some thinking about where the foam originates. So she went to the M.I.T. library and read about foam and surface chemistry and ran into Pattle's article on stable bubbles. She didn't like the idea of using a microscope to measure the diameter of bubbles in foam as a quantitative approach to research on edema so she asked Whittenberger about alternative methods. An Edgewood alumnus himself (from World War II), Whittenberger sent her to Clements to learn how to use a surface balance. Clements recalls that she probably didn't think much of his home-made balance but she went back to Boston where she and Mead made a better balance and went to work. Having a clinical traineeship at the Lying-in Hospital gave her access to lungs of babies who had just died of respiratory distress syndrome and, in no time, before cells could deteriorate, extracts of these lungs were in her surface balance.

Avery and Mead studied the lungs of individuals without "hyaline membrane disease": 35 were infants, 5 were children who died between 9 weeks and 4 years of age, and 4 were adults. The mean minimal surface tension (on compression of the surface film) was 7.6, 6.7, and 7.2 dyne per cm, respectively, in these three groups. The minimal surface tension of lung extracts from nine infants who died of hyaline membrane disease was 30.4 dynes per cm. They concluded (46):

> The results show that without exception the surface behavior of lung extracts of the nine infants with hyaline membrane disease was different from that of infants dying from other causes and the same as that of infants smaller than 1,200 gm. This suggests that the disease is associated with the absence or delayed appearance of some substance which in the normal subject renders the internal surface capable of attaining a low surface tension when lung volume is decreased.
>
> It is of interest to attempt to relate the results obtained to the pathogenesis of the disease. In all lungs with the first breath, large pressures are necessary to create an air-liquid interface. In this respect the normal lung would not differ from the lung without the surface active material since surface tension on extension of the surface is similar in both cases. Thereafter, during expiration, the alveolar surface of the normal lung would have diminished tension thus reducing the tendency of the air spaces to collapse. On the other hand, in a lung lacking this lining material, surface tension would tend to remain high during expiration: the air spaces would be unstable, and some would collapse. Once a sufficient number had closed, others would remain open inasmuch as the interpleural pressure at end-expiration would be sufficiently negative to prevent further closure. (46)

This explains clearly why the early explanation of atelectasis by Wilson and Farber (1933) based on cohesion (or stickiness) of the alveolar surfaces was an unsatisfactory one beyond the first inspiration of the newborn. Something had to be operating over a longer period of time to explain collapse of the lungs over and over again with each breath; it was the absence of surfactant instead of the presence of stickiness.

The measurements of Avery and Mead also provided the rationale for continuous positive airway pressure (CPAP) as a physiological method for managing the respiratory distress syndrome, though this was not accepted until the work of Gregory and associates in 1971.

Two asides here in connection with Avery's paper: *(1)* If you look at its credit line at the bottom of the first page, you will see that the work was supported by a special traineeship from the National Institute of Neurological Diseases and Blindness. I'm sure you're aware that a few congressmen (such as Senator—I forget his name, but he's the one who had transplant surgery [an unusual kind—external instead of internal]) scan the titles of projects to determine the appropriateness of support by a particular institute or indeed by any of the National Institutes of Health. In Avery's case, they might have been disturbed by finding that the Institute of Neurological Diseases and Blindness was supporting a youngster who wanted

to learn about the mechanical properties of the lungs. I can't find the original application (applications were then only two pages, with five lines allowed to describe the training plan) but I suspect that the body of the grant pointed out that the respiratory difficulties in the newborn might be due to damage to the respiratory center and hence be appropriate for support by a Neurological Institute. It's hard to judge a book (or a Retrospectroscope article) by its title and even harder to know the importance of a grant proposal by its title. *(2)* Inexplicable events occur in the transmission of scientific knowledge. In 1961, Jere Mead wrote his classic *Physiological Review* article, "Mechanical Properties of Lungs" (47). It included a ten-page scholarly discussion of pulmonary surface tension and 258 references; among these were a 1959 paper by Avery, Frank, and Gribetz and a 1960 paper by Avery but *not* the Avery and Mead paper of 1959 proving that the minimal surface tension of extracts was invariably high when prepared from the lungs of babies who died of hyaline membrane disease. Let this be a warning to those who depend upon review articles to find *every* important published article!

The Attack on Immature Lungs

Avery and Mead's discovery ended Phase 2 of this story. No longer were discoveries premature; each now reached receptive and perceptive basic and clinical scientists. But Phase 3 had now begun and a tremendous amount still had to be learned:

—what was the chemical composition of surfactant; would it be possible to synthesize it and deliver it directly to needy alveolar walls?

—what was the source of surfactant; where was it formed?

—what released surfactant; how did it get to the surface of alveoli and what regulated replenishment?

—when in fetal life did pulmonary surfactant appear? when was it present in sufficient concentration to permit normal respiration?

—could one learn whether it was present or absent in the fetus before the fetus became a newly-born baby?

—could one delay delivery if the lungs of the fetus were sure to be immature? could one hasten their maturation? or both?

—how could one best ventilate immature lungs that contain little or no surfactant—lungs that collapse after each successful inspiration?

(To be concluded)

Julius H. Comroe, Jr.

ACKNOWLEDGMENT

I thank Drs. M. E. Avery, J. A. Clements, R. E. Pattle, E. P. Radford, and K. M. Wilson for their kindness in reconstructing events of 20-25 years ago.

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MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ___ of ___

Form Approved OMB No 0910-0291 Expires 4 30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit sequence #

A. Patient information

1 Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4 Weight
___ lbs
or
___ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g. defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)
- ☐ death (mo/day/yr)
- ☐ life-threatening
- ☐ hospitalization – initial or prolonged
- ☐ disability
- ☐ congenital anomaly
- ☐ required intervention to prevent permanent impairment/damage
- ☐ other:

3 Date of event (mo/day/yr)

4 Date of this report (mo/day/yr)

5 Describe event or problem

6. Relevant tests/laboratory data, including dates

7 Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2 Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) (from/to / or best estimate)
#1
#2

4 Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6 Lot # (if known)
#1
#2

7 Exp. date (if known)
#1
#2

8 Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2. Type of device

3. Manufacturer name & address

4 Operator of device
- ☐ health professional
- ☐ lay user/patient
- ☐ other:

5. Expiration date (mo/day/yr)

6.
model #
catalog #
serial #
lot #
other #

7 If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10 Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1 Name & address

phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4 Also reported to
- ☐ manufacturer
- ☐ user facility
- ☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
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FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

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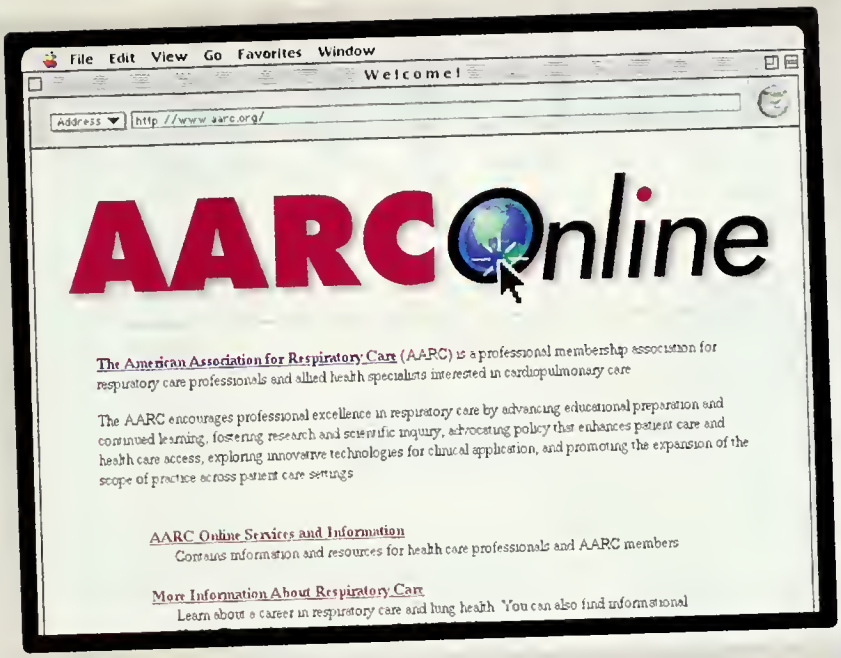
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AARCOnline
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MARKETPLACE

A

To celebrate its 50th year, the AARC is offering several anniversary items to commemorate this important milestone in respiratory care. Members who flip through this 16-page "catalog" will also find some RCP products and apparel at special discounted prices. There's no time like 1997 — the AARC's 50th year — to show your pride in the respiratory care profession.

B

50th Anniversary Poster

A This 17" by 22" poster is a collage of photos that showcase the last 50 years of the respiratory care profession.

Item AN18 $6 ($10 nonmembers)

Business Card Holder

B Black leatherette business card holder displays the 50th anniversary logo in gold. Great place to keep those business cards handy!

Item AN16 $5 ($7.50 nonmembers)

To Order by Credit Card or Purchase Order, Call the AARC at (972) 243-2272.

Anniversary Polo Shirt

C Walk around in style in this cotton knit polo-style shirt with embroidered 50th anniversary logo. This banded-sleeve shirt has a four-button placket and knit collar. Forest green only. Sizes L, XL, and XXL.

Item AN15 $45 ($60 nonmembers)

Brass Money Clip

D This brass money clip, etched with the 50th anniversary logo in dark blue, holds bills securely in your pocket. Classic souvenir of the AARC's 50 years.

Item AN10 $10 ($15 nonmembers)

D

C

E

Letter Opener

E Open your mail with ease. Letter opener features the 50th anniversary logo.

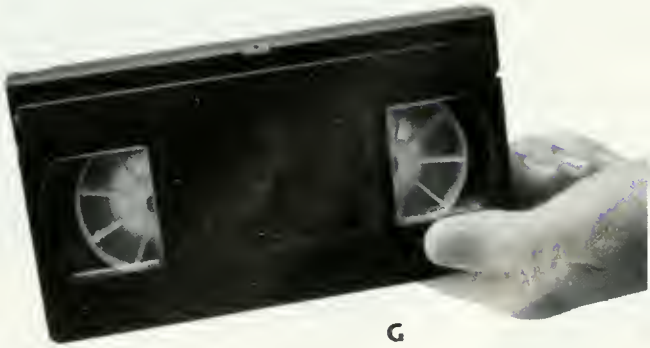
Item AN17 $1 ($1.50 nonmembers)

F

E-Cylinder Key Ring

F Carry your keys on this soft vinyl e-cylinder–shaped key ring.

Item AN14 $1 ($1.50 nonmembers)



G

50th Anniversary Video

G This 17-minute video traces the AARC's history since its birth in 1947 as the Inhalational Therapy Association.

Item AN19 $10 ($15 nonmembers)

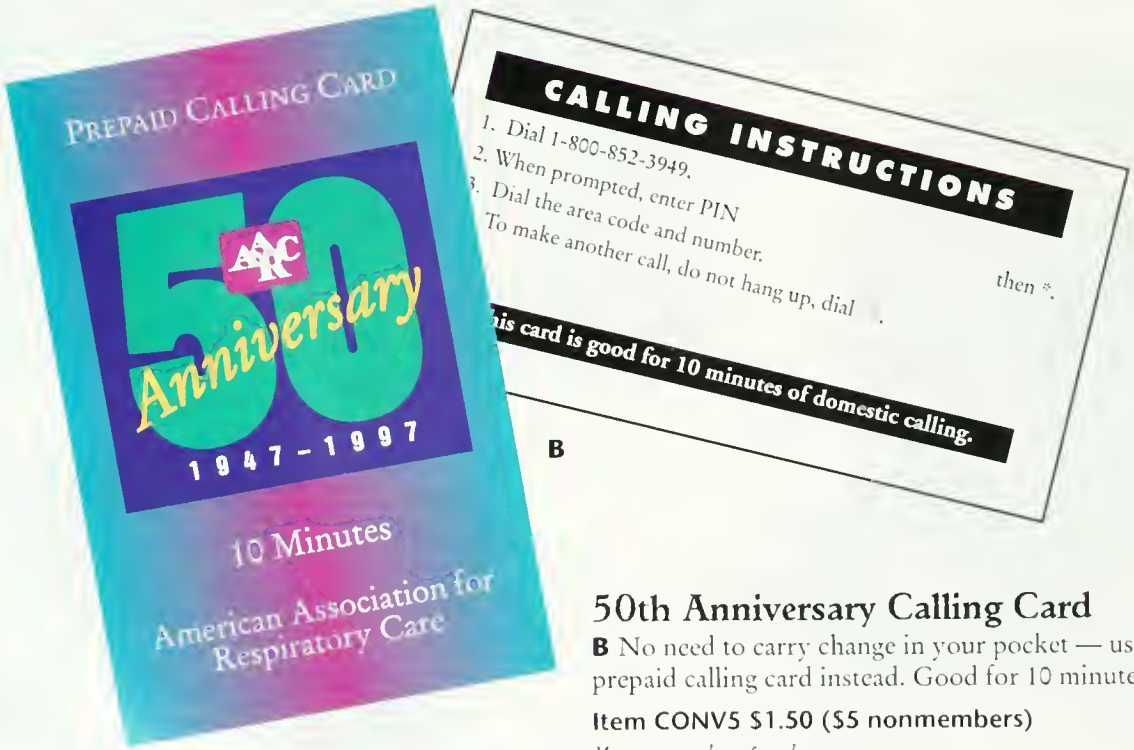
MARKETPLACE

A

50th Anniversary Jansport Backpack

A Nothing speaks quality and durability like a Jansport Backpack. Available in black with a brown-suede bottom and the AARC's 50th anniversary logo embroidered in gold. Roomy inside, with outside zipper pocket.

Item AN11 $39 ($50 nonmembers)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B

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B No need to carry change in your pocket — use this convenient prepaid calling card instead. Good for 10 minutes of domestic callin

Item CONV5 $1.50 ($5 nonmembers)

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C

Magic Mug

C Celebrate the AARC's 50 years with this unique, fun mug. Just pour in your favorite hot beverage and watch the 1947 logo on your magic mug fast-forward to 1997! Dishwasher use not recommended.

Item AN12 $10 ($12.50 nonmembers)

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D Use this soft foam mousepad with futuristic design and never forget the AARC's web address: http://www.aarc.org — it's right at your fingertips!

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E

Swiss-style Army Knife

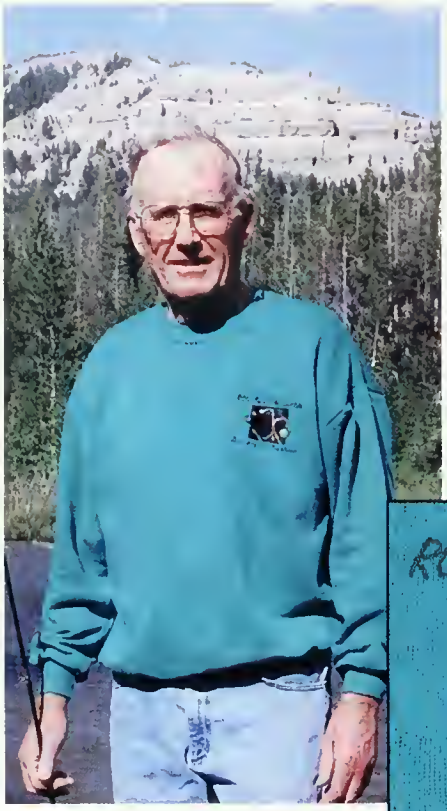
E This handy swiss-style army knife goes everywhere with you. Tucked inside is a knife, file, scissors, and toothpick. Comes in its own leather-like pouch.

Item AN13 $9 ($12 nonmembers)

D

Marketplace Bargains

Many Prices Marked Down. Hurry, Some Quantities Are Limited.

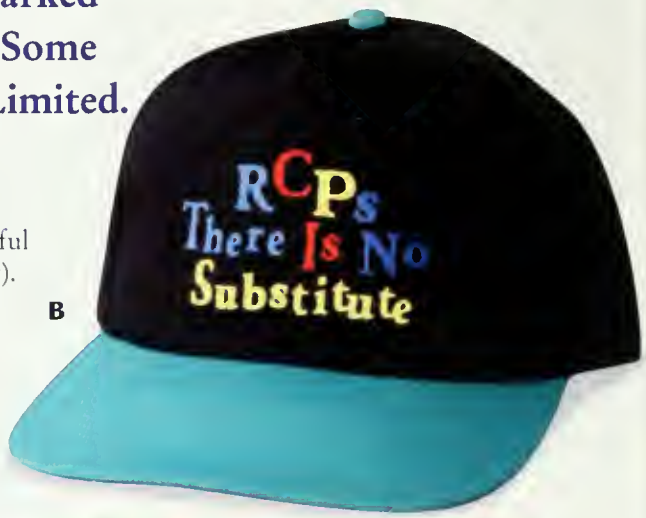


A

RC Sweatshirt

A 50/50 blend with colorful embroidered logo (below). Sizes M, L, XL, XXL.

Item R19 $20 ($25 nonmembers)
Add $2.50 for XXL.



B

RC Cap

B One size fits all.

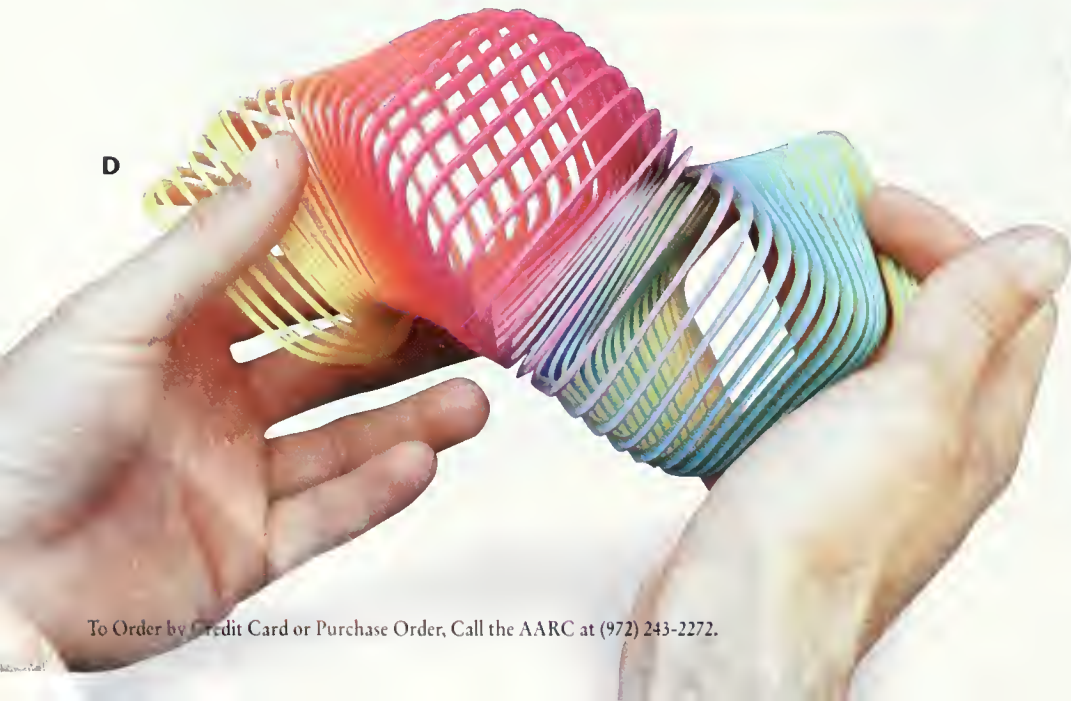
Item R17 $5.25 ($7.25 nonmembers)

C

Pull-Apart Key Ring

C Metal key ring lets you separate your car keys from your home keys.

Item R90 $1 ($2 nonmembers)



D

The Pick-Me-Up

D Use as a stress-reliever, pencil holder, message sorter, toy, etc.

Item R11 $2 ($4 nonmembers)

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E

Balloons

E Package of 100 9" round latex.

Item R1 $10 ($12 nonmembers)

Foil Balloons

F 18" silver mylar balloons. Package of 3.

Item R2 $2.50 ($5 nonmembers)

For safety's sake, do not release foil balloons near power lines.

F

G

Fanny Pack

G Features 3 zippered pockets. Adjustable waistband for a comfortable fit.

Item R3 $4.75 ($6.50 nonmembers)

H

Colorful T-Shirt

H 50/50 white cotton/polyester blend. Sizes M, L, XL, XXL.

Item R16 $6 ($8 nonmembers)

Add $2.50 for XXL.

Table Tents

I Package of 25.

Item R14 $4.25 ($6.50 nonmembers)

Display Banner

J 1' by 8'.

Item R4 $7 ($12 nonmembers)

Pencils

K Package of 50.

Item R5 $16 ($25 nonmembers)

I

J

K

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MARKETPLACE

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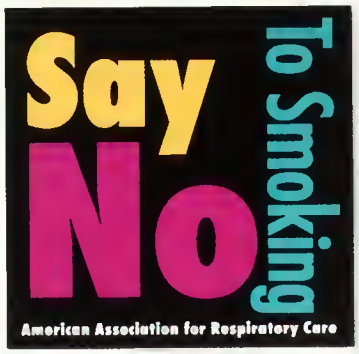
No-Smoking Buttons

A Package of 20. Safety-back pin.

Item R6 $6 ($10 nonmembers)

B



No-Smoking Stickers

B Package of 50.

Item PR32 $2.25
($4.25 nonmembers)

Headless Matches

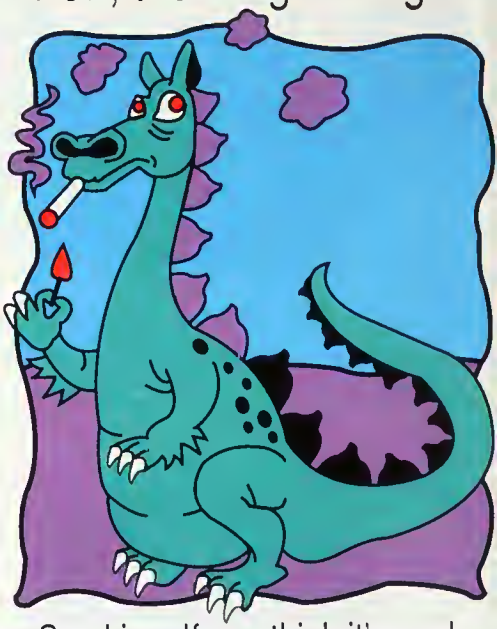
C Box of 50.

Item R9 $2.25 ($4.25 nonmembers)

C

D

Puff, the Tragic Dragon



Smoking: If you think it's cool, it's only your imagination.

American Association for Respiratory Care

Dragon Poster

D 12" by 16."

Item R44 $2.50 ($5 nonmembers)

Non-Smoking Fans

E Package of 25.

Item R8 $7.50 ($11 nonmembers)

Stop-Sign Bookmarks

F Package of 25.

Item R12 $4.75
($7 nonmembers)

E

F

Mini Flyers

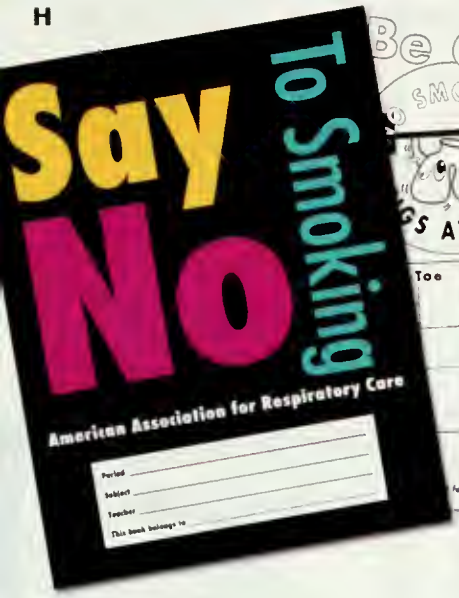
G Package of 10.

Item R10 $3.50
($7 nonmembers)

G

To Order by Credit Card or Purchase Order, Call the AARC at (972) 243-2272.

H



I

J

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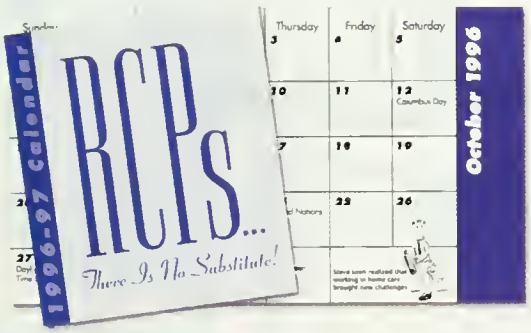

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K 15-month calendar through December 1997.

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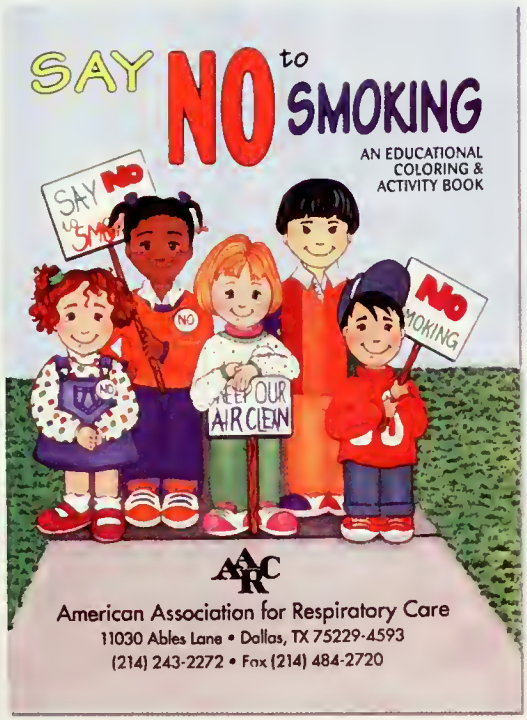
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American Association for Respiratory Care

N

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MARKETPLACE

When did these symptoms start?

¿Cuando empe. estos síntom.

Page 48

A

B

Eye Shields

A Reusable frames come in assorted colors. Package of 25 disposable lenses and 5 reusable frames.

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Spanish Phrase Book

B Flashcard-style notebook to communicate with Spanish-speaking patients. Fits in lab coat pocket.

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AARC Patches

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Milk-caps

D Kids can add to their POG™ collection with these Cool Cat milk-caps. Package of 20 (10 of each design).

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G Complements the framed AARC Statement above.

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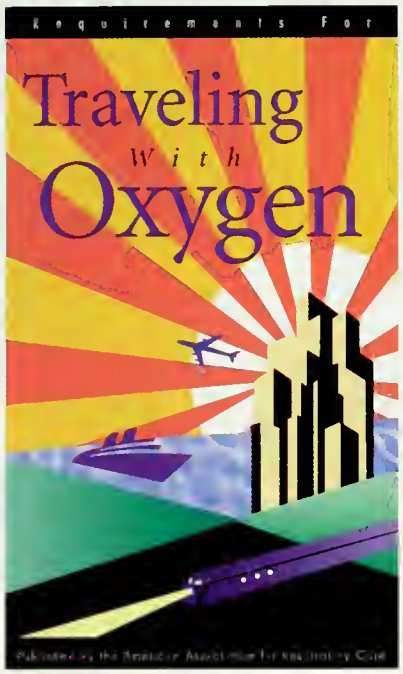

Requirements For Traveling With Oxygen

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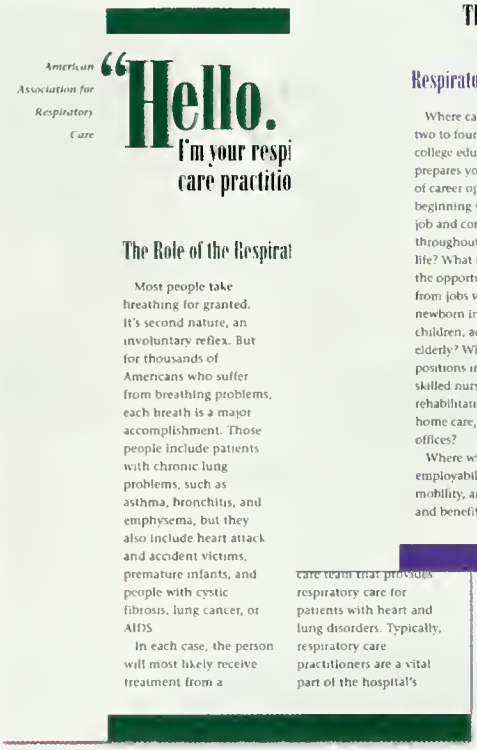
American Association for Respiratory Care
"Hello.
I'm your respi
care practitio
The Role of the Respirat
Most people take breathing for granted. It's second nature, an involuntary reflex. But for thousands of Americans who suffer from breathing problems, each breath is a major accomplishment. Those people include patients with chronic lung problems, such as asthma, bronchitis, and emphysema, but they also include heart attack and accident victims, premature infants, and people with cystic fibrosis, lung cancer, or AIDS.
In each case, the person will most likely receive treatment from a
care team that provides respiratory care for patients with heart and lung disorders. Typically, respiratory care practitioners are a vital part of the hospital's

"Hello. I'm Your Respiratory Care Practitioner"

I This brochure tells your patients who you are and what you do. Package of 50.

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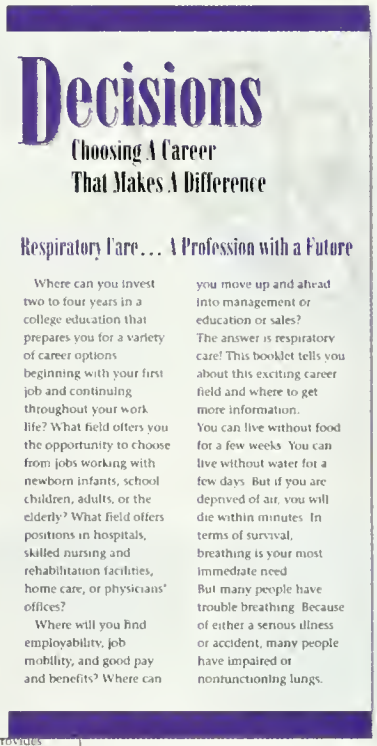
J


American Association for Respiratory Care
Decisions
Choosing A Career
That Makes A Difference
Respiratory Care... A Profession with a Future
Where can you invest two to four years in a college education that prepares you for a variety of career options beginning with your first job and continuing throughout your work life? What field offers you the opportunity to choose from jobs working with newborn infants, school children, adults, or the elderly? What field offers positions in hospitals, skilled nursing and rehabilitation facilities, home care, or physicians' offices?
Where will you find employability, job mobility, and good pay and benefits? Where can you move up and ahead into management or education or sales?
The answer is respiratory care! This booklet tells you about this exciting career field and where to get more information.
You can live without food for a few weeks. You can live without water for a few days. But if you are deprived of air, you will die within minutes. In terms of survival, breathing is your most immediate need.
But many people have trouble breathing. Because of either a serious illness or accident, many people have impaired or nonfunctioning lungs.

"Decisions: Choosing A Career That Makes A Difference"

J Discusses opportunities and benefits in the respiratory care field. Package of 50.

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K 18" by 24" attention-grabbing poster.

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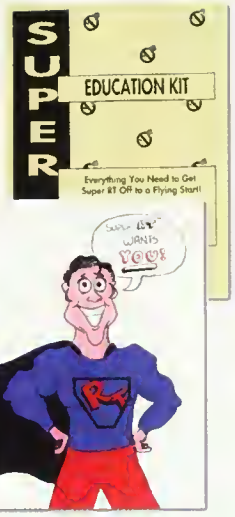

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Order Item PR7XL for an XL shirt

A Each kit contains a Super RT T-shirt (available in L or XL), three puppets, three different scripts of short plays to illustrate the dangers of smoking, 30 activity books, 30 non-smoking fans, 50 Smokebusters stickers, 30 Smokebusters bags, and a lesson plan.

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C

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B Features a colorful wrap-around design.

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C Enameled with safety clasp.

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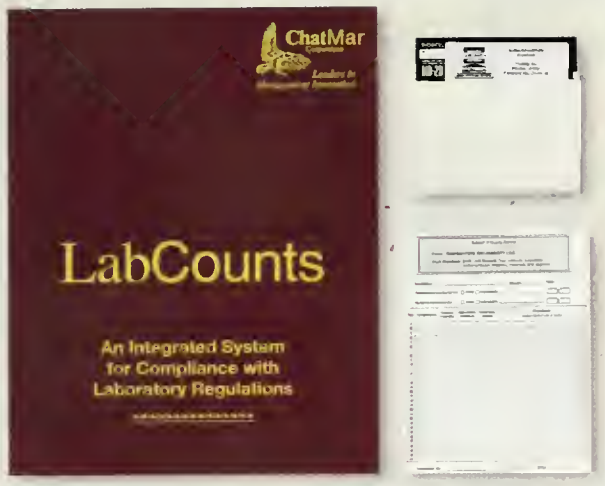

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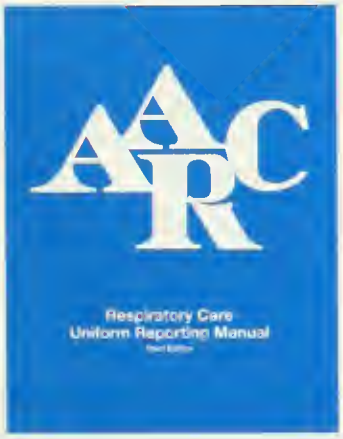

The AARC Human Resources Survey: A Study of Respiratory Care Human Resources in Hospitals

68 pages, 66 tables.

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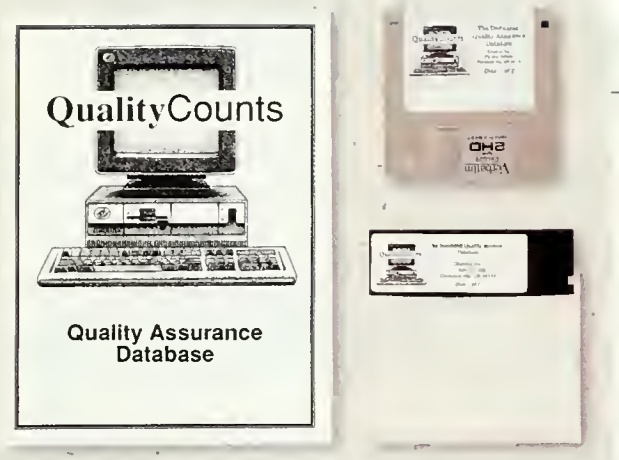

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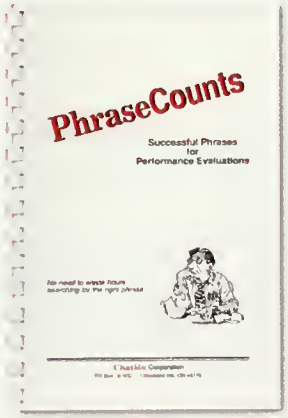

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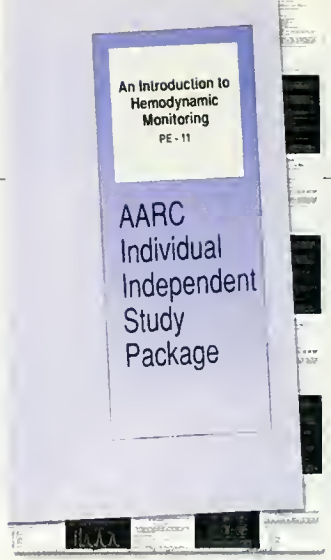
Individual Independent Study Packages (IISPs) Help Practitioners Expand Their Respiratory Care Knowledge



An Introduction to Hemodynamic Monitoring

This IISP teaches you to recognize the types of equipment used for hemodynamic measurements. It also instructs in position of the pressure transducer, identification of pressure waveforms, and measurement of cardiac output. Discusses anatomic normal pressure values, oxygen saturation values, and the two major determinants of cardiac output.

Item PE11

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation

After completing this IISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.

Item CP4

Tracheal Intubation II: Routes of Intubation

Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.

Item CP5

Pulmonary Edema

Reviews normal anatomy and physiology, and highlights the pathophysiologic processes of pulmonary edema and the clinical presentation of the abnormality. Also identifies patients at risk and appropriate interventions available to diminish the impact of the condition.

Item CP10

Clinical Practice

Chest Tubes and Pleural Drainage

Item CP3

Tracheal Intubation III: Equipment Procedures for Intubation

Item CP6

Respiratory Management of Neuromuscular Crisis

Item CP7

Respiratory Management of Flail Chest

Item CP8

Respiratory Management of Head Trauma

Item CP9

Clinical Science

Transpulmonary Pressure Changes in Breathing

Item CS5

Lung Mechanics

Item CS6

Practical Application of Gas Laws

Item CS7

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Item CS13

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Bronchodilators II: Anti-Cholinergics and Xanthines

Item CS15

Calculation and Preparation of Respiratory Medications

Item CS16

Microbiology for Respiratory Therapy: A Review of Microbial Growth and Cross-Contamination

Item CS17

Classification of Mechanical Ventilators I

Item CS18

Classification of Mechanical Ventilators II

Item CS19

Classification of Mechanical Ventilators III

Item CS20

Ventilation/Perfusion Relationships in Health and Disease

Item CS21

Neonatal

Recognition and Stabilization of the Premature Infant in Respiratory Distress

Item NN1

Patient Evaluation

Pulmonary Function Assessment I: Basic Screening Studies

Item PE3

Pulmonary Function Assessment II: Bedside Studies

Item PE4

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies

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Body Plethysmography

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Nicotine Dependency Evaluation and Treatment

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Bedside Counseling of the Hospitalized Smoker

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Health Tips Fact Sheets

B These one-page fact sheets help you share your knowledge and expertise on important issues in respiratory health with the community. Pad of 50.

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Topics Include:

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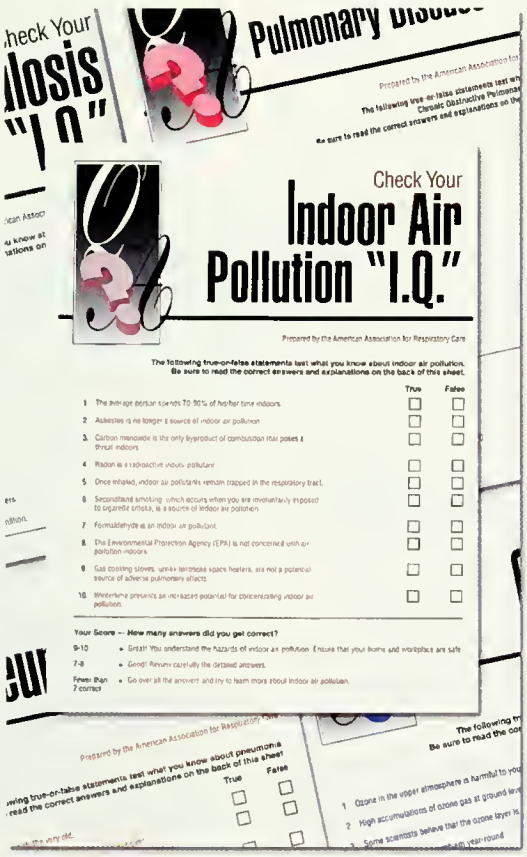
Secondhand Smoke ..... **Item R24**
Eating Right: Tips for the COPD Patient ..... **Item R25**
Dealing with Allergies ..... **Item R30**
Minimizing the Effects of Outdoor Air Pollution ..... **Item R31**
Living with Asthma ..... **Item R32**
The Process of Quitting Smoking ..... **Item R33**
Coping with Indoor Air Pollution ..... **Item R34**
Exercising Safely with COPD ..... **Item R35**
Lessening the Effects of COPD ..... **Item R36**
Cystic Fibrosis ..... **Item R37**
Tuberculosis ..... **Item R38**
The Effects of Ozone Pollution ..... **Item R39**
Lessening the Effects of Sleep Apnea ..... **Item R40**



Quiz Sheets

A Inpatients and outpatients alike will find these "IQ Tests" challenging and informative. These true-and-false tests feature detailed answers on a variety of subjects vital to respiratory care. Pad of 50.

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Topics Include:

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Check Your COPD "IQ" ..... **Item R61**
Check Your Cystic Fibrosis "IQ" ..... **Item R62**
Check Your Indoor Air Pollution "IQ" ..... **Item R63**
Check Your Outdoor Air Pollution "IQ" ..... **Item R64**
Check Your Pneumonia "IQ" ..... **Item R65**
Check Your Secondhand Smoke "IQ" ..... **Item R66**
Check Your Smoking "IQ" ..... **Item R67**
Check Your Tuberculosis "IQ" ..... **Item R68**
Check Your Ozone "IQ" ..... **Item R69**
Check Your Sleep Apnea "IQ" ..... **Item R70**

"Professor's Rounds in Respiratory Care" Video Series

NEW! Prevention and Management of Ventilator-Induced Lung Injury

Featuring David J. Pierson, MD, and Richard D. Branson, RRT. This presentation reviews the data from both animal experiments and clinical experience, and describes our current concepts of the pathophysiology of ventilator-induced lung injury. It also covers how to identify patients at increased risk of barotrauma, measures to decrease the likelihood of this complication, and the approach to management of bronchopleural fistula in the ventilated patient.

Item VC52

NEW! The Multidisciplinary Team Approach and Respiratory Care

Featuring Kevin L. Shrake, MA, RRT, FAC HE, and Sam P. Giordano, MBA, RRT. This program focuses on what respiratory care practitioners must do to demonstrate value and create opportunity and how they can develop a professional skills inventory that enhances value and solidifies job security. Also presented are restructuring concepts that focus on developing the multiskilled practitioner capable of functioning on a variety of teams throughout the continuum of care. Advice is also provided for managers who want to position themselves as "clinical executives" who will guide the future of integrated health care delivery systems.

Item VC53

NEW! Emergency Respiratory Care: The Respiratory Care Practitioner's Role

Featuring Charles G. Durbin, Jr., MD, FCCM, and Richard D. Branson, RRT. Reviews some of the new approaches to emergency care, "tricks of the trade" in airway maintenance, and changes in the ACLS philosophy and treatment protocols. Methods to increase participation in the hospital emergency response team and the obstacles the team must overcome are presented.

Item VC54

NEW! Noninvasive Mechanical Ventilation: Its Role in Acute and Chronic Ventilatory Failure

Featuring Nicholas S. Hill, MD, and Richard D. Branson, RRT. This presentation reviews the history of noninvasive ventilation, describes modalities of noninvasive ventilation currently available, and discusses the advantages and disadvantages of the various types. Results of studies on acute and chronic respiratory failure are reviewed, and acceptable indications for use of noninvasive ventilation are described. Considerations for selecting appropriate patients, and techniques of initiation and monitoring of noninvasive ventilation, are also discussed.

Item VC55

NEW! Organizing a Respiratory Care Department Without Walls

Featuring John R. Walton, MHA, MBA, RRT, FAC HE, and Sam P. Giordano, MBA, RRT. This videotape discusses how to establish respiratory services in the post-acute setting using examples of a successful respiratory care service established in a subacute skilled nursing facility. Also provides insight on how the successful respiratory care service must be designed to follow the flow of the patient from the acute care hospital through alternate sites.

Item VC56

NEW! Shortening Length of Stay on Ventilators and in Hospitals

Featuring Neil R. MacIntyre, MD, and Richard D. Branson, RRT. This videotape focuses on the reasons why patients remain ventilator dependent and the disease specific factors of ventilatory muscle overload, neurological dysfunction, and gas exchange difficulties. Maximizing therapy for the underlying cardiorespiratory disease is also presented as a component in shortening the patient's length of stay. The tape also discusses ventilation management issues and complications such as pneumothorax, imposed work from ventilator settings and equipment, aspiration pneumonia, nutrition, and fluid.

Item VC57

NEW! Managed Care and Its Implication for Respiratory Care

Featuring Patrick J. Dunne, MEd, RRT, and Sam P. Giordano, MBA, RRT. Examines the opportunities and implications for respiratory care delivery of services in managed care. Discussion focuses on how multiskilling makes respiratory care more valuable in the managed care environment. Also discusses the skills needed to prosper in managed care and how managed care affects site selection, patient education, patient monitoring, and the utilization of services.

Item VC58

NEW! Management of Life-Threatening Asthma

Featuring David J. Pierson, MD, and Richard D. Branson, RRT. This program helps the viewer recognize the patient who is at risk of a life-threatening event and teaches how to assess the severity of the episode. It also discusses the general approach to the management of life-threatening asthma and reviews the current strategies for mechanical ventilation of the patient who has life-threatening asthma.

Item VC61

NEW! Managing the Ventilator: What and When

Featuring Dean R. Hess, PhD, RRT, and Richard D. Branson, RRT. The viewer learns initial ventilator setup and selection of appropriate settings. The program covers monitoring the patient, responding to clinical data with appropriate adjustments, the techniques for effective secretion clearance and infection control, and recognizing who is ready to wean.

Item VC62

NEW! Managing Demand for Respiratory Care Services

Featuring James K. Stoller, MD, and Sam P. Giordano, MBA, RRT. Participants learn how managing demand for services ties into organizational values and how to identify the major components of a successful utilization control program. The program will help respiratory care practitioners identify potential demand management roles for RCPs outside of the acute care facility.

Item VC63

NEW! Capturing Respiratory Services Outside the Hospital Part I: Home Care

Featuring Alan B. Sapoznik, MS, RRT, and Sam P. Giordano, MBA, RRT. This program describes the predicted future trends in home care and identifies the incentives for reimbursement for professional respiratory services in the home under capitated reimbursement. Also explains the value added by respiratory care practitioners in the delivery of home health services.

Item VC64

NEW! Theory and Application of Neonatal Ventilation: What, When, and Why

Featuring Rob Chatburn, RRT, and Richard D. Branson, RRT. The participant learns why neonates need ventilation, how to assess and monitor the neonate on the ventilator, and how to recognize the neonate in respiratory failure. Also provided is instruction in the strategies for minimizing iatrogenic lung damage, with a summary of the application of the new modes of ventilation and respiratory support in neonates.

Item VC65

NEW! Capturing Respiratory Services Outside the Hospital Part II: Subacute Care

Featuring Kevin A. Cornish, RRT, and Sam P. Giordano, MBA, RRT. This presentation explains how respiratory services are reimbursed by Medicare. Further discussion is presented on how to differentiate between the different types of subacute care and what skills are needed by respiratory care practitioners to practice successfully in subacute care.

Item VC66

NEW! Airway Management: Tricks of the Trade

Featuring Charles G. Durbin, Jr., MD, FCCM, and David J. Pierson, MD. With this program, viewers learn how to manage the obstructed airway and how to determine when an artificial airway is necessary. Also presented are the methods of assessing endotracheal tube positioning, monitoring the intubated patient, and determining when extubation is feasible.

Item VC67

Pressure Support During Mechanical Ventilation

Item VC21

Hemodynamic Monitoring

Item VC22

Weaning from Mechanical Ventilation

Item VC23

Pulmonary Rehabilitation

Item VC24

Aerosol Administration

Item VC25

Prevention of Postoperative Atelectasis and Pneumonia

Item VC26

Patient-Focused Care

Item VC31

Application of Positive Airway Pressure Without Intubation

Item VC32

Therapist-Driven Protocols in Respiratory Care

Item VC33

Monitoring Oxygenation in the Critically Ill Patient

Item VC34

Pulmonary Function Testing: When, Why, and What

Item VC35

Unconventional Methods for Adult Oxygenation and Ventilation Support

Item VC36

Tuberculosis: Implications for Patients and Practitioners

Item VC41

Therapist-Driven Protocols: Implementation

Item VC42

New Strategies for Asthma Management

Item VC43

Blood Gas and Related Measurements: Laboratory Versus Bedside Devices

Item VC44

Hospital Operational Restructuring and Respiratory Care

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The New Ventilator Management: Permissive Hypercapnia and Other Variations on Conventional Mechanical Ventilation

Item VC46

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Please Note: Videotapes do not qualify viewer for CRCE credit.

"Current Concepts in Respiratory Care" Video Series

Pulmonary Rehabilitation
By John E. Hodgkin, MD. Provides an overview of the sequence for pulmonary rehabilitation. Discusses aerosol therapy, IPPB, oxygen therapy, and chest physiotherapy in the treatment of COPD.
60 minutes.
Item VT7

Sleep Apnea
By Alan K. Pierce, MD. Explains how sleep stages are related to respiratory patterns and blood gas values in both normal and abnormal subjects.
60 minutes.
Item VT11

Practical Management of ARDS
By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape, and its clinical features are described, including incidence of risk factors and clinical predictors.
60 minutes.
Item VT16

Pressure Support Ventilation
By Neil MacIntyre, MD. This presentation defines and describes the physiologial considerations attributed to PSV and the clinical situations when PSV may be useful.
0 minutes.
Item VT17

Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak
By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function, potential problems associated with chest tubes stripping, and techniques for maintaining gas exchange with air leaks. 60 minutes.
Item VT21

Clinical Use of the Swan-Ganz Catheter
By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter placement and the situations in which SGC placement are useful are described in this video.
60 minutes.
Item VT22

Fetal Lung Development
By Charles Rosenfeld, MD. Examines the four anatomical phases of fetal lung development and discusses the biochemical development of the fetal lung through gestation.
60 minutes.
Item VT23

Theory and Application of Neonatal Ventilation
By Robert Chatburn, RRT. Presents a well-organized and systemic approach for managing mechanical ventilation. 60 minutes.
Item VT25

Tissue Oxygen Delivery
By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. 60 minutes.
Item VT26

Managing the Work of Breathing During Mechanical Ventilation
By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation.
60 minutes.
Item VT27

Pressure Support Update
By Neil MacIntyre, MD. Reviews pressure support ventilation and how it is used. Explains waveforms for airway pressure, flow, and volume.
60 minutes.
Item VT28

The Hospitalized COPD Patient: 10 Commandments for the Clinician
By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. 60 minutes.
Item VT29

Monitoring Respiratory Mechanics During Mechanical Ventilation
By Robert L. Chatburn, RRT.

Explains how physical and mathematical models are developed and applied and how they are incorporated into ventilator design to provide estimates of mechanics.
60 minutes.
Item VT30

ARDS Review
By Tony Dal Nogare, MD. Discusses the latest developments in risk factors and treatment.
60 minutes.
Item VT31

Sleep Disorders
By Brian Foresman, DO. A discussion of the physiology of sleep and the kinds of respiratory and nonrespiratory sleep disorders seen in the hospital. 60 minutes.
Item VT32

Uses and Abuses of Noninvasive Monitors in Respiratory Care
By Dean Hess, MEd, RRT. Presents a discussion of how much noninvasive monitoring is needed, proof of false positives, its financial impact, and its future.
54 minutes.
Item VT33

Nutrition and Respiratory Care
By Rich Branson, RRT. Explains the relationships and interactions of malnutrition on ventilatory drive, respiratory muscles, lung structure, and immunity.
60 minutes.
Item VT34

Smoking Cessation: Intervention Techniques for the Respiratory Care Practitioner
By Kathleen A. Smalky, MD, MPH. Covers four major aspects of smoking cessation — the impact of smoking on illness and mortality, behavioral components, current cessation programs, and effective intervention. 43 minutes.
Item VT35

Hospital Acquired Pneumonia
By Galen B. Toews, MD. Provides information on the epidemiology, pathogenesis, and management of nosocomial pulmonary infections.
52 minutes.
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Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

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Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

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Article in a journal carrying pagination throughout volume:

1. Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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Oliver Wendell Holmes said that once stretched by a new idea, the human mind never regains its original dimensions. For me, being a member of the American Association for Respiratory Care has greatly contributed to the expanding of my mind by helping me achieve a rewarding career in respiratory care.

I joined the AARC as a student member in 1974 because my professors expected it. However, as I began to receive new ideas, I came to realize that being a member of the AARC was fundamental to becoming a true professional. Through active membership, I have been able to stay abreast of the ever-increasing information, technology, and reforms in respiratory care. I have also been able to contribute ideas of my own by participating in AARC conferences, moderating the annual OPEN FORUM, and serving as a House of Delegates Officer and as a RESPIRATORY CARE Editorial Board member. Today, I encourage my students to join the AARC as part of their becoming a respiratory care professional. RESPIRATORY CARE, *AARC Times*, Clinical Practice Guidelines, conference proceedings, and networking with other health professionals are excellent resources for all respiratory care practitioners.

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- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
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- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

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City _____

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Phone No. (_____) _____

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- ☐ 4 years
- ☐ Other, specify _____

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- ☐ Associate Degree
- ☐ Bachelor's Degree
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- ☐ Doctorate Degree

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- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
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Credentials

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- ☐ CRTT
- ☐ Physician
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- ☐ RPFT
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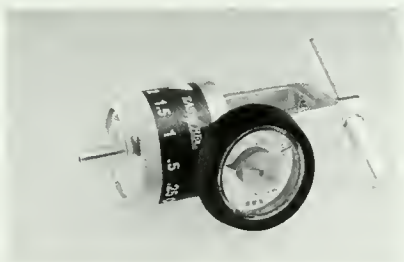
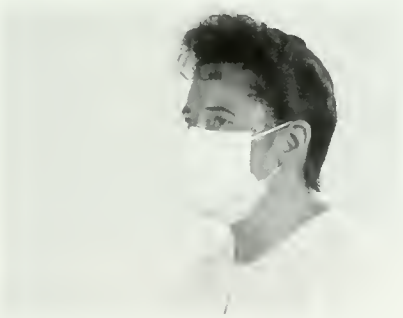
New Products & Services

TAPE. Hy-Tape® Corporation launches its Original Pink Tape® in a new 1/4 in. size. Hy-Tape is a water resistant tape created for use with neonatal patients. The tape remains intact even in the presence of moisture, has a zinc oxide-based adhesive, aids in the reduction of epidermal stripping, and reduces the risk of skin injuries, claims the company. The tape also allows for endotracheal tubes to be secured without compromising skin integrity. Details are available; circle Reader Service Number 160.



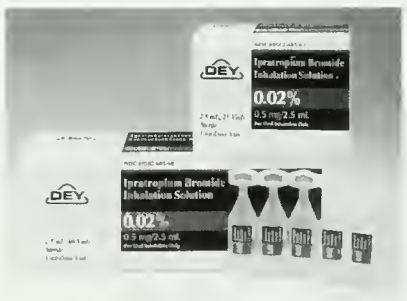
OXYGEN REGULATOR. Precision Medical Inc introduces the Easy/Dial Reg—a small-dial, lightweight oxygen regulator. The dial measures 3.5 in., weighs 4.4 oz., and features an easy-to-read gauge. The compact design uses a new offset gauge and a multispring technology. Large numbers and an arrow indicate the flow settings, which can be set via the adjustment knob. A 'click' mechanism signals proper oxygen flow setting. The regulator also features a rubber boot that shields the gauge from damage. And, the regulator is available in 0.25-8 L/min and 0.5-15 L/min flow models. Circle Reader Service Number 161 for details from Precision Medical.

CPAP SYSTEMS. Respironics Inc launches two new continuous positive airway pressure (CPAP) systems—Virtuoso™ and Aria™. The Virtuoso, an automatically adjusting continuous positive airway pressure system, is specifically designed for the treatment of adult obstructive sleep apnea (OSA) and monitors a patient's airway while delivering appropriate pressure for therapy. Furthermore, the system tracks the patient's changing needs and adjusts to changing sleeping positions and sleep stages. Respironics claims that the system is quiet and easy to use. The Aria is also used in OSA and is developed to be quiet, compact, and easy for patients to use. The unit delivers a wide range of pressures and is tolerant of most mask leaks. Details about both CPAP systems and other units in the Great Performers™ Series offered by Respironics are available, circle Reader Service Number 162.



FACE MASK. The Medical Face Mask is offered by the United Medical Enterprises Inc. The mask features 99% filtration material, meets NFPA 702 Class 1 flammability requirements, is tear resistant, has a soft, aluminum-enclosed nose piece, and meets U.S. Food & Drug Administration regulations, the company says. In addition, the mask is safe to use and reduces the possibility of allergic reactions. The mask contains no fiberglass or latex filter media and traps submicron-sized particles. To obtain more information, circle Reader Service Number 163.

POINT-OF-CARE SYSTEM UPDATE. Mallinckrodt Sensor Systems announces the addition of an information management system to its GEM® Systems R.O.I. Program for point-of-care testing. According to Mallinckrodt, the system collects, verifies, transmits, and archives point-of-care data. The system can be programed to include monthly fixed costs for equipment and consumables that are directly tied to actual hospital usage. This approach allows hospitals to more effectively manage fixed and variable costs. For details about the system, circle Reader Service Number 164.



GENERIC INHALATION SOLUTION. DEY Laboratories now supplies a generic alternative to Atrovent® Inhalation Solution—Ipratropium Bromide Inhalation Solution. Ipratropium Bromide is AN rated and is offered in both 25- and 60-vial packages. The solution, according to DEY literature, is an anticholinergic bronchodilator for the maintenance treatment of bronchospasm in COPD, and is not for use in the initial treatment of acute episodes of bronchospasm. For more information regarding indications and contraindications, dosing requirements, or clinical applications, circle Reader Service Number 165.

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Calendar of Events

AARC & AFFILIATES

May 15–17—Colorado Society
"Mission Possible," in Estes Park, Colorado.
Contact: Jill Brown at (800) 547-3511.

May 20—AARC Live Video Conference
"JCAHO Problematic Areas for Respiratory Care Services," Part 3 of AARC's 1997 "Professor's Rounds in Respiratory Care," from 12:30 to 2 PM Eastern Time.
CRCE: 1 credit hour.
Contact: AARC at (972) 243-2272.

May 20–21—Connecticut Society
Super Symposium, "Respiratory Care: The Magic Is in Your Hands," at the Four Points Sheraton, Waterbury, Connecticut.
Contact: Nancy Apruzzese, Director, Respiratory Care Services, (860) 224-5264.

May 21—AARC Live Telephone Question-and-Answer Session
View "Waveform Analysis and Interpretation," Part 2 of AARC's 1997 "Professor's Rounds in Respiratory Care," and participate in a live telephone question-and-answer session from 12:30 to 1 PM Eastern Time.
CRCE: 1 credit hour.
Contact: To receive the videotape and register for the teleconference, call the AARC at (972) 243-2272.

June 4–6—New Jersey, New York State, & Pennsylvania Societies
10th Annual Managers/Educators Rocking Chair Conference at the Winter Clove Inn and Conference Center, Round Top, New York.
The $250 registration fee includes conference registration, 2 nights' lodging, meals, and gratuities.
Contact: Ken Wyka in New Jersey at (201) 288-3959, Joseph Sorbello in New York at (315) 464-6872, or John Hughes in Pennsylvania at (717) 291-8208.

June 9—AARC Live Telephone Question-and-Answer Session
View "JCAHO Problematic Areas for Respiratory Care Services," Part 3 of AARC's 1997 "Professor's Rounds" series and participate in a live telephone question-and-answer session from 12:30 to 1 PM Eastern Time
CRCE: 1 credit hour.
Contact: To receive the videotape and register for the teleconference, call the AARC at (972) 243-2272.

June 11–13—Illinois Society
29th annual convention, "Healthcare's Multi-Skilled Players," at the Indian Lakes Resort, Bloomingdale, Illinois
Contact: Larry Dastych at (847) 618-7710.

June 11–13—Texas Society
Annual convention, "The Star of Texas," at the Marriott Riverwalk Hotel, San Antonio, Texas. The Management Training Institute seminar is also scheduled.
Contact: TSRC at (972) 680-2454.

August 6–8—New Mexico Society
Annual convention, "At the Hop," at the Albuquerque Convention Center, Albuquerque, New Mexico.
Contact: Brooke Patterson, VA Hospital, (505) 265-1711, ext. 4998.

August 21—Ohio Society
Annual Critical Care Seminar, "Rock-n-Roll and Respiratory Care," hosted by MetroHealth Medical Center, Cleveland, Ohio.
Contact: Bob Messenger at (216) 887-3226 or e-mail bmessenger@mhmc.org.

OTHER MEETINGS

September 25–26—INFOCARE
Second Annual Neonatal/Pediatric Respiratory/Critical Care Symposium at the Sheraton Fiesta Beach Resort, South Padre Island, Texas
Contact: Ralph Miller, INFOCARE, 350 San Roman Rd, Los Fresnos TX 78566, (210) 233-5645 or fax (210) 233-1010.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

International Sepsis Forum

The International Sepsis Forum has been formed to improve awareness, understanding, and clinical management of sepsis. The group intends to address: early diagnosis and identification of patients at risk; best use of current management strategies; new and emerging therapies; ethical and economic issues; and standardization of training. Interested physicians and health care providers are encouraged to join these discussions by contacting Pharos Healthcare Communications at (203) 622-6769.

ASTM Standards via Fax

The American Society for Testing and Materials (ASTM) now offers WEBFAXX, a new option available at http://www.astm.org in the "Search for Standards" area. Copies of the standards, once requested, can be sent within 10 minutes to any fax machine. The copies cost $0.75 in the United States, Canada, and Mexico and $1.50 in all other countries.

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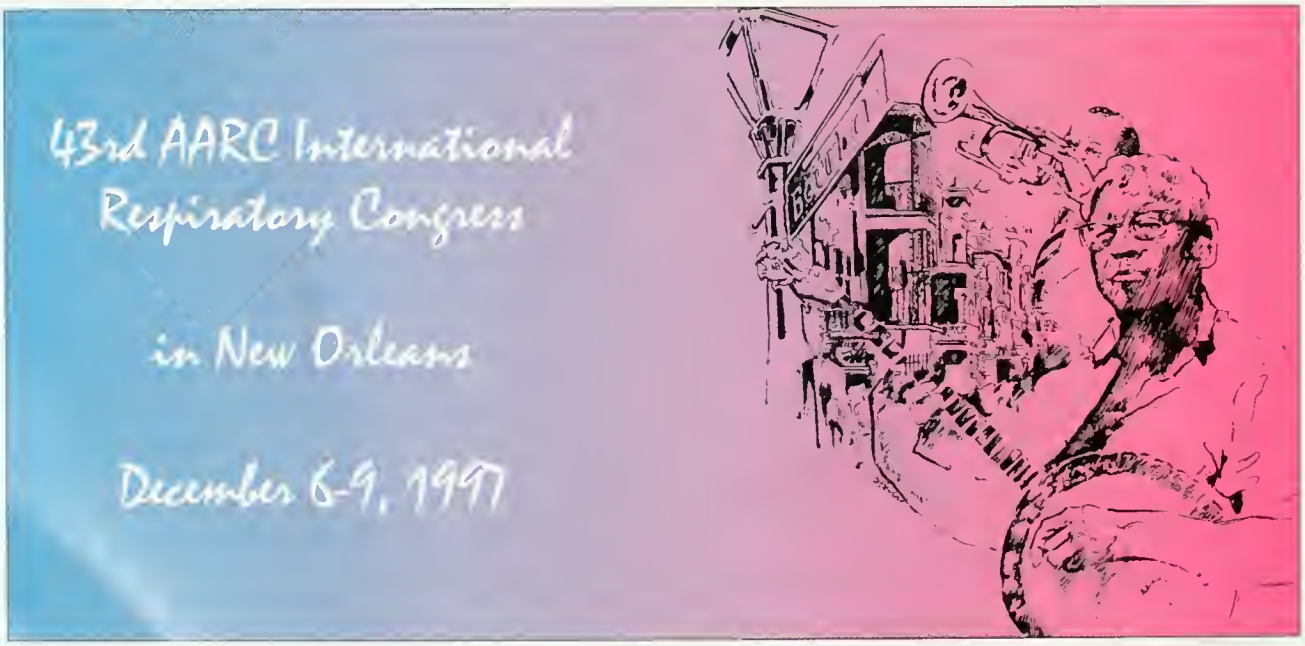

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Examination	Examination Date	Examination Fee	
CRTT Examination	March 8, 1997	$ 100	(new applicant)
	Application Deadline: January 1, 1997	60	(reapplicant)
	July 12, 1997	100	(new applicant)
	Application Deadline: May 1, 1997	60	(reapplicant)
	November 8, 1997	100	(new applicant)
	Application Deadline: September 1, 1997	60	(reapplicant)
RRT Examination	June 7, 1997	100	Written only (new applicant)
	Application Deadline: February 1, 1997	60	Written only (reapplicant)
		110	CSE only (all applicants)
	December 6, 1997	210	Both (new applicant)
	Application Deadline: August 1, 1997	170	Both (reapplicant)
CPFT Examination	June 7, 1997	110	(new applicant)
	Application Deadline: April 1, 1997	80	(reapplicant)
RPFT Examination	December 6, 1997	160	(new applicant)
	Application Deadline: September 1, 1997	130	(reapplicant)
Perinatal/Pediatric Respiratory Care Specialty Examination	March 8, 1997	160	(new applicant)
	Application Deadline: November 1, 1996	130	(reapplicant)

For information about other services or fees, write to the
National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, email nbrc-info@nbrc.org.

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6. ❑ Distributor

II. Department
A. ❑ Respiratory Care
B. ❑ Cardiopulmonary
C. ❑ Subacute Care
D. ❑ Home Care

III. Specialty
1. ❑ Clinician
2. ❑ Perinatal/Pediatrics
3. ❑ Critical Care
4. ❑ Research
5. ❑ Subacute Care
6. ❑ Diagnostics/ Pulmonary Function
7. ❑ Management
8. ❑ Home Care
9. ❑ Rehabilitation
10. ❑ Education

IV. Position
A ❑ Department Head
B. ❑ Chief Therapist
C. ❑ Supervisor
D. ❑ Staff Therapist/ Technician
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